Strategies for monitoring and training strength and power in elite rugby union players.

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ABSTRACT

Rugby union requires high levels of strength and power in order to support the physical requirements of the game. The competitive structure of rugby union in the English premiership places limitations on the time available for players' physical development. The aim of this thesis was to analyse the scope and magnitude of strength and power adaptation potential, whilst identifying effective training strategies to support physical development in professional rugby union players. Chapter 3 monitored lower limb strength and power during the different phases of a professional season. This study demonstrated moderate beneficial increases in all physical capacities over a full season whilst pre and mid-season training cycles represent the greatest opportunity for strength and power enhancement. Chapter 4 assessed the efficacy of complex training performed during a mid-season performance phase and found meaningful increases in selected measures of power whilst maximum strength was maintained. Chapter 5 assessed the impact of pre-conditioning exercise mode selection (cycling or weightlifting) when designing complex training interventions and reported highly individualised response patterns in measures of lower and upper body performance. Chapter 5 also demonstrated no clear support for the short-term effects of elevated free-testosterone on local and systemic muscle performance. Chapter 6 investigated the effects that manipulating work interval duration has on fast muscle activity and power during high intensity interval training (HIT). This study reported greater accumulative power responses and fast muscle activation in selected muscles when shorter work interval durations were prescribed. In summary, scope for physical development exists in professional rugby union players. Complex training may be an efficient in-season training method for power development. Hormonal response patterns represent unpredictable markers of acute and chronic improvements in local and systemic muscle performance. Finally, the endurance potential of fast muscle groups may benefit from HIT protocols designed with shorter work interval durations.

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CHAPTER 1: INTRODUCTION

1.1 Research context and background literature

Rugby union is classified as a collision based field sport that is intermittent in nature and requires high levels of strength, power, speed and fitness (Roberts et al., 2008). Competition play is punctuated with short maximal accelerations, high impact collisions and a high frequency of static and dynamic exertion efforts when competing to gain or maintain ball possession (Smart et al., 2014, Deutsch et al., 2007). These demands require players to be proficient in high force and velocity-dominant movements (Cross et al., 2014). Consequently, the development of both leg strength and power is critical if the kinematics of effective acceleration is to be developed (Barr et al., 2014a). Equally, strength development is an important factor that supports improvements in two of the key variables which discriminate elite from sub-elite rugby union performance, sprint momentum and collision effectiveness (Barr et al., 2014b, Crewther et al., 2009c). Sprint capacity has also been reported to be a direct key performance indicator of successful phase and team outcomes during match-play, with initial acceleration measures demonstrating correlations to line breaks, tackle breaks and tries scored in professional rugby union (Smart et al., 2014).

Developing the physical qualities to support match-play performance in a professional squad environment poses numerous difficulties. The competing physiological and sport-specific requirements of elite rugby may compromise the development of strength, power, speed and repeated power production due to the interference of concurrent training effects (Hawley, 2009). The multiple in-week frequency of tactical and technical training and the weekly match play demands of professional rugby union can also place considerable time limitations on physical conditioning (Appleby et al., 2012). In accordance with the law of diminished returns, developing strength and power in elite athletes is also progressively harder to achieve (Cormie et al., 2010b). Consequently, identifying training strategies which facilitate effective and efficient conditioning of elite rugby players is of critical importance if physical characteristics are to be enhanced.

A large proportion of training for rugby union is spent developing an athlete's ability to express, co-ordinate and repeat contractile muscle force. This in turn may result in improved functional performance and fatigue resistance in game related activities such as acceleration, scrummaging, tackling, rucking and mauling. Three common training modalities employed to improve physical capacities in rugby union players include:

i) Resistance training for the development of peak force via morphological, architectural and neurological adaptation in skeletal muscle (Andersen and Aagaard, 2010);

ii) Power training for the development of multi-joint dynamic performance through increases in the synchronisation, firing frequency and co-ordination of high threshold motor units (Cormie et al., 2011a);

iii) High-intensity interval training to promote high threshold muscle fibre fatigue resistance via increases in cardiorespiratory and muscle peripheral oxidative capacity (Buchheit and Laursen, 2013b).

There is little scientific data detailing how the acute and accumulative demands of a competitive season of English premiership rugby union impacts upon the application and efficacy of various resistance and power training modalities. Identifying how resistance and power training can be programmed and monitored in conjunction with weekly match play and rugby specific training demands throughout the various phases of a season is required if effective training interventions are to be implemented. The initial experimental research in this thesis (Chapter 3) will look to identify the nature, scope and magnitude of chronic adaptations in strength and power during pre and in-season training cycles during a full competitive season of professional rugby union in the English premiership

Investigating specific training strategies which have the potential to improve strength, power and power repeatability is important if practical training guidelines are to be developed within the context of the holistic professional rugby environment. Currently, only a small body of research has focussed on identifying training techniques which improve physical performance for elite rugby code athletes (Argus et al., 2009, Baker and Newton, 2006, Comfort et al., 2012).

Complex training is a technique commonly employed in applied team sport settings as a means of developing strength and power and its effectiveness will be assessed in Chapter 4 and Chapter 5. Complex training involves the execution of heavy weightlifting (1 to 5 repetitions) or maximal isometric contraction followed, after a period of recovery, by the execution of biomechanically similar power or plyometric exercise (i.e. a complex set) (Docherty D, 2004). The premise on which complex training is based assumes that the explosive capability of a muscle is acutely enhanced after it has just been subjected to a maximal or near maximal contraction. This phenomenon has been referred to as post-activation potential (PAP). Subsequently, complex training is believed to enhance the acute expression and chronic development of power as a consequence of performing previous high intensity strength based pre-conditioning activity. This training modality may also provide a practically efficient

strategy for integrating strength and power development into a time-limited athletic environment (Lloyd and Deutsch, 2008).

Complex training has yet to be validated as an effective training method for improving strength and or power within an elite rugby environment. Assessing a chronic complex training strategy will provide a greater understanding of the relative importance of this training mode and provide information to aid the programme design characteristics which support any associated improvements in strength, power and speed. Analysing how the acute manipulation of a complex set impacts on the subsequent expression of power may provide information as to the prescription of pre-conditioning exercise modalities which best facilitate elevated power output.

Hormone monitoring has also been proposed to inform training adaptation potential in response to various athletic development strategies (Ahtiainen et al., 2003, Crewther et al., 2011b). Biological alterations in testosterone and cortisol in response to strength training have been shown to mediate long-term changes in skeletal muscle protein metabolism, muscle cross-sectional area and consequently peak force production (Hakkinen et al., 1985, Hakkinen, 1989, Kraemer et al., 1999, Ahtiainen et al., 2003). Acute increases in the dynamic expression of force as a consequence of physical pre-conditioning strategies have also been associated with changes in testosterone concentrations (Obminski et al., 1998, Crewther and Cook, 2010, Cook and Crewther, 2012). However, the value of monitoring hormone response patterns to resistance training in elite rugby union players has yet to be fully established. Manipulating the short and long-term hormonal environment via specific complex training strategies could promote an elevated training response which may support functional improvements in strength and power. Monitoring the acute hormone response to pre-conditioning routines will also allow for associations between hormones and dynamic performance to be identified. These response pathways will be analysed in Chapter 4 and Chapter 5. This research may inform guidelines for the training methods which facilitate the manipulation of hormones in order to improve workout performance and adaptation potential in elite rugby athletes.

Rugby union players also require an elevated capacity to repeat high muscle forces under conditions of fatigue generated in response to the high intensity intermittent efforts of match play (Roberts et al., 2008). Specific fitness strategies, such as short work duration high-intensity interval training (HIT) (i.e. work interval durations lasting <30 s), require small time commitments and total training volumes whilst also providing the adaptive potential to improve skeletal muscle aerobic power (Buchheit, 2014). However, little is known on how best to structure the programme design characteristics of HIT to ensure the exercise intensity facilitates the engagement of high threshold motor units. This concept will be the focus of Chapter 6. Investigating the effect that manipulating work and relief interval duration has on

muscle activation and total exercise power will provide new insights as to how changes in programme design can elevate the acute performance response and fast muscle fibre adaptation potential of a HIT session.

For the transfer and adoption of training theories to be effective within any sport, evidence must show that these principles are effective and practical in a real world setting (Bishop, 2008). To this end, this PhD was designed to further improve and evolve applied performance and physiological knowledge regarding the efficacy of specific strength and conditioning strategies commonly used by coaches in elite rugby union environments.

1.2 Purpose of research

The overall aim of this research programme will be to identify acute and chronic training modalities which promote strength and power development in elite rugby union players within the context of a professional English premiership season, in order to provide practical recommendations to inform the design of training programmes.

The specific objectives of this thesis will be to:

1) Monitor strength and power development together with retention and decay potential over pre-season, mid-season and end of season training phases in the English premiership;
2) Investigate the effects of implementing a complex training programme on measures of strength, power and speed over an in-season training cycle;
3) Examine salivary steroid hormonal response patterns to complex and strength training strategies over an in-season training cycle;
4) Identify the acute potentiating effects of two physical pre-conditioning strategies on subsequent measures of power;
5) Investigate associations between exercise induced alterations in blood concentrations of steroid hormones and power;
6) Identify the effect of work interval duration manipulation on fast muscle fibre activity and power during short HIT exercise.

1.4 Structure of thesis

This thesis is initially comprised of a literature review with a focus on four key themes (Figure 1.1). The first section of the review (2.1) will identify the importance of strength and power training for elite athletic performance by exploring the morphological and neurological factors which underpin muscle force production and coordination. Section 2.2 will review the physiological mechanisms responsible for post activation potential and the associated

application of complex training as a tool for developing strength and power within athletic groups. Section 2.3 will identify current evidence detailing the importance of hormones in mediating long-term neuromuscular adaptation and short-term neuromuscular functioning for elite athletes in response to resistance training. The last review section (2.4) will explore the physiological and performance adaptations associated with short duration HIT whilst also examining the programme design characteristics that influence training prescription. Four experimental studies will then be performed which look to monitor and develop strength and power over long-term training periods (Chapter 3) and in response to chronic (Chapter 4) and acute training strategies (Chapter 5 and Chapter 6) (Figure 1.1). The first experimental study (Chapter 3) is a longitudinal tracking study identifying strength and power development, maintenance and decay potential in professional rugby union players competing throughout pre and in-season training phases (objective 1). The next two studies will look to characterise the chronic and acute application of complex training and hormone monitoring. Chapter 4 will monitor performance adaptations in strength, power and speed in response to a complex training intervention performed during an in-season training cycle whilst also report any training associated alterations in acute and chronic hormones (objectives 2 and 3). Chapter 5 will concentrate on acute strategies which potentiate muscle power through the manipulation of pre-conditioning exercise modality (objective 4). This study will also attempt to identify if hormonal manipulation can enhance acute local and systemic muscle performance in an applied training setting (objective 5). Chapter 6 will analyse acute training strategies that support repeated power development in elite rugby players by identifying the work to relief interval durations that support greater fast muscle fibre activity and power outputs during short HIT (objective 6).

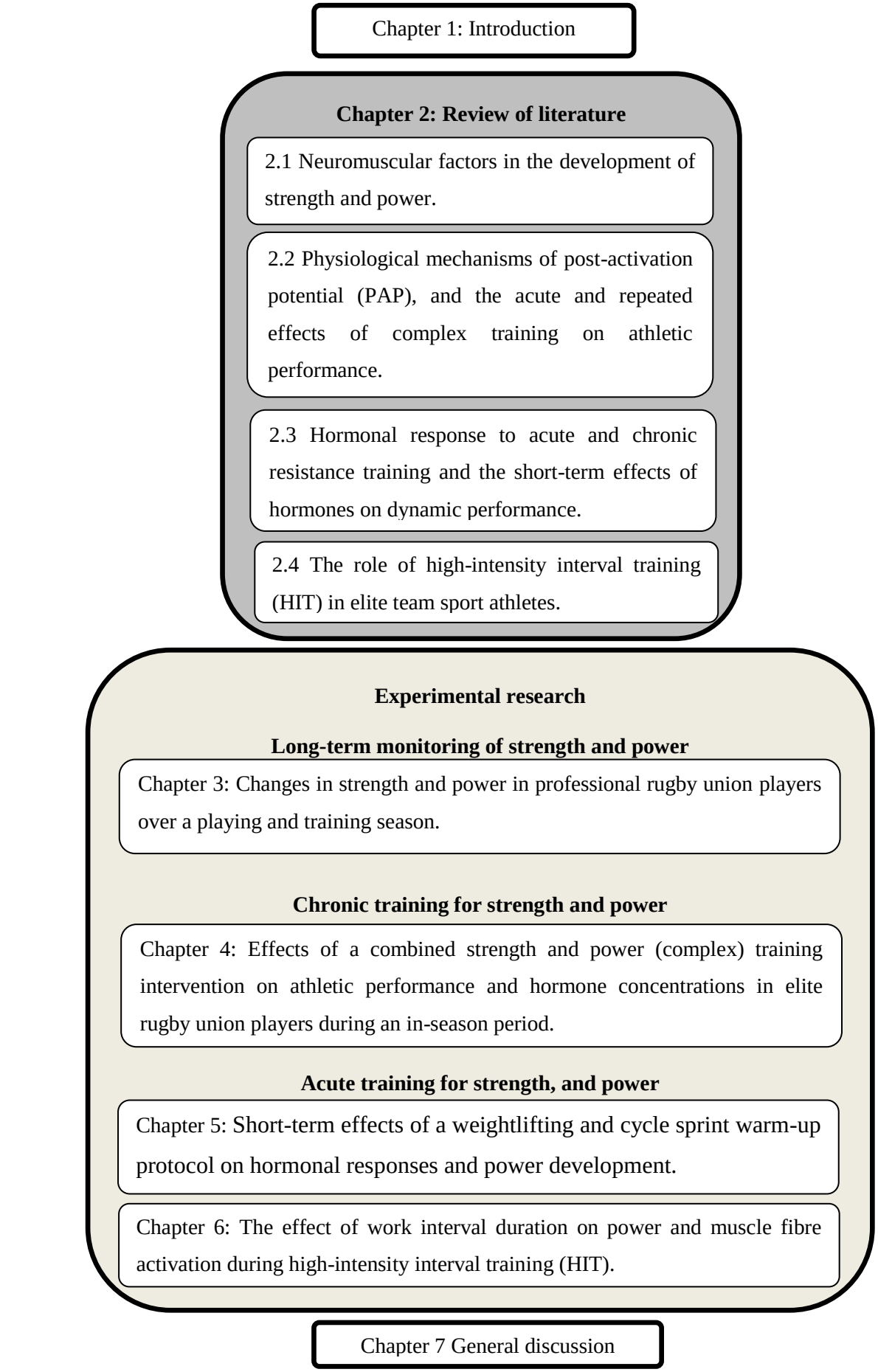

Figure 1.1 Thesis structure.

CHAPTER 2: REVIEW OF LITERATURE

2.1 Neuromuscular factors in the development of strength and power

2.1.1 Introduction

The neuromuscular system controls mechanical muscle function for daily activities as well as athletic tasks. The adaptive plasticity of the neuromuscular system in response to a variety of training stimuli can evoke marked changes in muscle morphology and nervous system function. Cumulatively, these changes contribute to significant increases in maximal muscle contractile force and power production. Strength can be defined as a system's ability to generate force, whilst power is the product of force multiplied by velocity (Stone et al., 2003). The association between strength and power appears therefore to be highly robust. Recent evidence demonstrates that increases in maximum strength alone can cause increases in power production (Cormie et al., 2010b, Hanson et al., 2009), whilst athletes who are stronger demonstrate greater improvements in ballistic performance after a period of power training (Cormie et al., 2010a). Consequently, the development of training programmes that effectively enhance muscular strength and or power must specifically target positive alterations in the neuromuscular factors which underpin force production.

The morphological profile of an athlete's muscle contractile properties demonstrates one area where strength training can enhance an athlete's ability to express force. Adaptive increases in muscle cross sectional area via heavy strength training have been shown to mediate increases in maximal power production (Shoepe et al., 2003). Increases in muscle pennation angle and fascicle length have also demonstrated plasticity to adaptation through resistance training and are believed to increase both muscle force generating capacity and muscle contractile velocity (Cormie et al., 2011a). Whilst the expression of muscle force appears to improve in conjunction with changes in muscle architecture, the ability of the nervous system to orchestrate muscle activation for specific kinematic conditions is also a pivotal characteristic that facilitates contraction capacity. Consequently, maximal strength training has been shown to be effective at altering the recruitment, co-ordination, and timing of motor unit activation strategies (Cormie et al., 2010b, Cormie et al., 2011c). Development of these neurological qualities may therefore provide a base of force generation that must then be transferred and tuned into functional gains in mechanical power output through ballistic training protocols.

Section 2.1 will look to provide insight into the potential adaptive mechanisms that control nervous system function for the expression of strength and power. This information may improve understanding into the acute and chronic role of the nervous system during high force

and velocity requiring movements. Section 2.1.2 will explore the neuromuscular association between strength and power and outline how this relationship is influenced by athletic training status. Section 2.1.3 will define the neuromuscular factors that dictate the acute function and chronic adaptability of the nervous system for muscle contraction.

2.1.2 Strength and power association

The ability to generate maximal force whilst executing rapid complex motor skills is of paramount importance for successful athletic performance. Consequently, coaches and sport scientists strive to develop effective and efficient training programmes which develop power by targeting the mechanisms which underlie adaptation. One of the fundamental principles dictating power production is an athlete's strength status. As power is the product of force multiplied by velocity, it appears logical that an athlete cannot possess a high level of power without first being relatively strong (Cormie et al., 2011c). Cross sectional analysis has shown that athletes who possess greater strength levels within homogenous groups display markedly superior power production capabilities than those with lower strength levels (Baker and Newton, 2008). This relationship is demonstrated to an even greater degree when the superior strength and power capabilities in elite (stronger) athletes are observed against sub-elite (weaker) athletes (Cormie et al., 2010b, Stone et al., 2003). Developing the neuromuscular basis for power production appears therefore to initially rely on the adaptations associated with heavy strength training.

A large body of evidence exists which demonstrates the positive effect of an isolated strength training programme on the development of power and speed (Comfort et al., 2012, Cormie et al., 2010b). In particular, Cormie et al. (2010a) found that weaker athletes reported greater adaptive benefits across a wider spectrum of the force-velocity continuum (e.g. increased peak force, rate of force development, power and speed) in response to a strength training protocol, whereas ballistic training elicited adaptations specific to the kinematics of the exercise stimulus (e.g. rate of force development and power). Cormie et al. (2010a) concluded that the wider improvements in athletic performance associated with strength training make this form of training a more effective modality for relatively weaker/untrained individuals. These athletic improvements may be associated with positive adaptations in the neuromuscular characteristics which form the basis for force production. For example, improved muscle architecture (cross sectional area of type II muscle fibre, increased pennation angle, increase fascicle length) (Folland and Williams, 2007, Hakkinen et al., 2002b, Shoepe et al., 2003) and neural drive (motor unit recruitment, rate coding, inter and intra muscular coordination) (Cormie et al., 2010b, Hakkinen, 1989, Aagaard et al., 2002) are all factors which have demonstrated adaptability to training in weaker athletes. As a result, these athletes may exhibit

a shift in the force velocity relationship so that the force generated by muscle would be greater for any given velocity of contraction (Stone et al., 2003) (Figure 2.1).

While strength is a basic quality that influences maximum power production, the degree of this adaptive influence diminishes when the athlete maintains a high level of force production. It has been reported that as strength is increased the window for adaptation for further strength and power improvements decreases (Cormie et al., 2011c). Strength training for elite athletes may therefore be governed by the principles of diminished return (Appleby et al., 2012). As a result, stronger athletes may enhance athletic performance to a greater degree by focussing on transferring the magnitude and timing of force production via velocity focused exercise programmes such as maximum power training. Training studies into the effects of power training have found that stronger athletes demonstrate greater magnitudes of neuromuscular adaptation and performance gains after ballistic training when compared with weaker athletes (Cormie et al., 2010b). However, whilst power training may be more beneficial for strength trained athletes, training strategies for these athletic groups must incorporate protocols which maintain the neuromuscular characteristics of strength. If strength is not maintained then the subsequent decrements in force production may negatively affect the ability of stronger athletes to adapt to power training (Cormie et al., 2010b). It appears that elite athletes require varied training structures that incorporate a mix of muscle force and velocity outputs. This may include strength maintenance exercises in conjunction with ballistic training at a variety of different external loads and muscle contraction speeds.

In conclusion, evidence indicates that the neuromuscular factors which dictate strength also underpin power production. As a result of this relationship, the current strength level of an athlete will always dictate the upper limit of their potential to generate power. When designing training programmes, attention must primarily be paid to the athletes baseline strength status. Improvements in muscle function in weaker untrained athletes will therefore be achieved in a non-specific manner via the use of strength training alone; however, stronger athletes will require a multifaceted approach which targets a variety of areas across the force velocity continuum. The next section will discuss in more detail the neuromuscular characteristics susceptible to training adaptation which underpin the expression of both strength and power.

2.1.3 The neuromuscular basis of force production

The relationship between strength and power may be explained by the interrelated factors that influence the biological basis of force production. The following review will analyse the morphological and neurological factors that dictate force production capacity in response to strength and power training protocols. These include increases in muscle cross sectional area,

fascicle length, and pennation angle as well as central nervous system control of motor unit recruitment, firing frequency, muscular synchronisation and inter-muscular coordination.

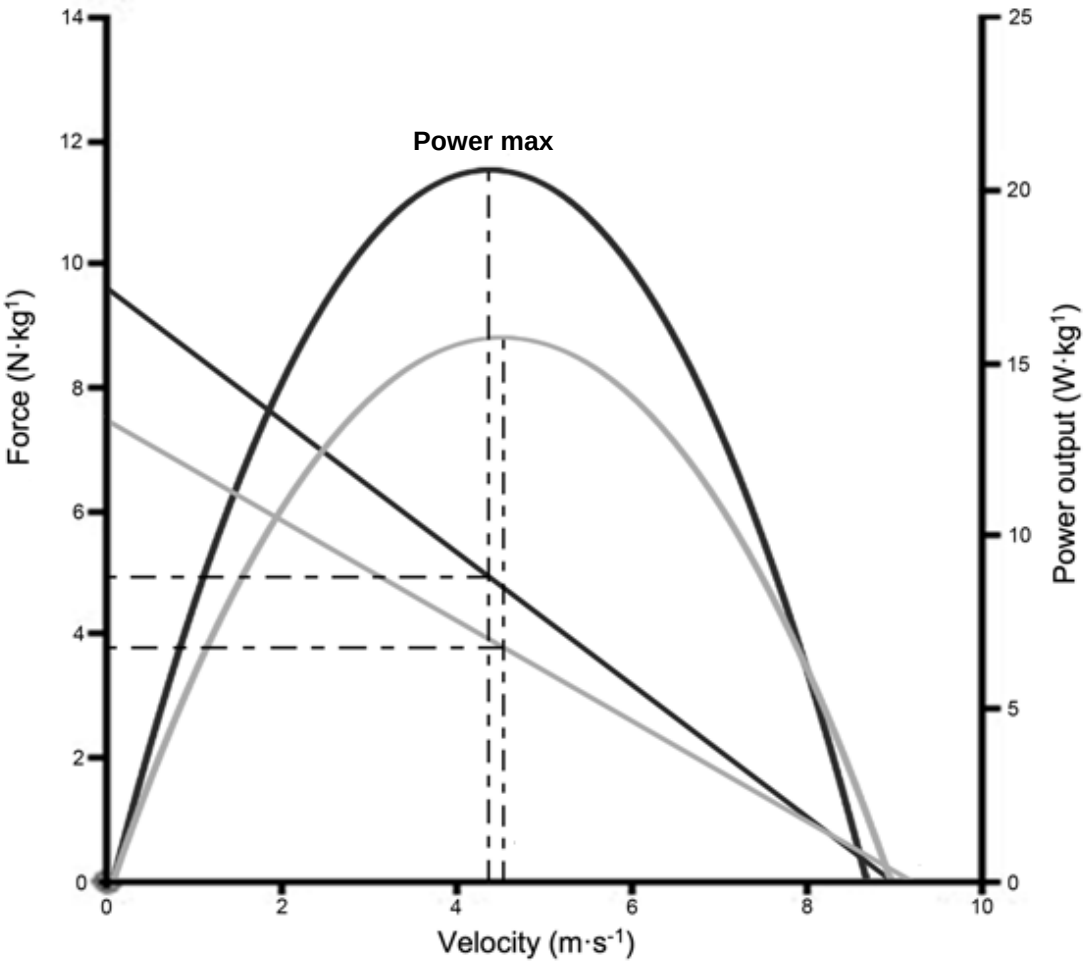

Figure 2.1 The effects of strength training on the force-velocity-power relationship of muscle. Grey line = pre strength training; black line = post strength training. Dashed lines represent the effect increasing force production capacity has on power production. Adapted from Cormie et al. (2011a).

Cross sectional area

The maximal force generated by a single muscle fibre is directly proportional to its cross sectional area (Cormie et al., 2011b). As power is heavily influenced by maximal force production, it appears logical to that a muscle fibre with greater cross sectional can therefore generate higher power outputs (Hanson et al., 2009). Greater cross sectional area in both slow and fast muscle groups has been demonstrated to account for the differences in peak power produced by resistance trained athletes in comparison to sedentary males (Shoepe et al., 2003). Strong associations have also been reported between knee extension maximum force and quadriceps cross sectional area in men (r = 0.71) and women (r = 0.76) (Jones et al., 1989).

Power production is also influenced by the composition of certain muscle fibre types. Elevated power outputs have been documented in muscle groups with a higher percentage cross sectional area of Type II fibres in comparison to muscle groups with a higher cross sectional area of Type I fibres (Tihanyi et al., 1982). Subsequently, hypertrophic adaptations in Type II muscle fibre is a primary outcome associated with heavy strength training. Repeated bouts of high intensity training result in the application of mechanical tension through an active muscle. The consequence of high muscle loading initiated through resistance training protocols causes mechanosensory regulation of muscle protein synthesis through intracellular signalling pathways (Egan and Zierath, 2013). The adaptive outcome in response to high mechanically loaded resistance training is muscle hypertrophy (i.e. increases in the size and number of myofibrils within a muscle fibre [Cormie et al., 2011b]). Consequently, the potential to increase maximal power through greater muscle cross sectional area relies on the appropriate application of a heavy strength training stimulus to induce the initiation of muscle hypertrophic adaptive response pathways.

Fascicle length

The maximum velocity of contraction within a muscle fibre is directly proportional to its length (Blazevich et al., 2009). Consequently, longer muscle fibres (i.e. a greater number of sarcomeres in series) would have a greater contractile velocity capacity (Cormie et al., 2011b). As velocity of contraction is heavily associated with power production, longer fascicle lengths would appear beneficial for power orientated athletes. Indeed, cross sectional analysis has demonstrated sprinters to have significantly longer fascicle lengths in the vastus lateralis, and gastrocnemius in comparison to long distance runners and untrained controls (Abe et al., 2001). Consequently, developing exercise strategies which enhance muscle fibre length would appear beneficial for athletes looking to develop power. Currently limited research exists detailing the influence of certain training modalities on adaptations in fascicle length. Heavy resistance training has been reported to increase fascicle length (Alegre et al., 2006, Blazevich et al., 2009), equally sprinting and jumping interventions have also resulted in fascicle length adaptations (Blazevich et al., 2003). However, not all of these adaptations supported improvements in dynamic performance. One study in particular reported decreases in early rate of force development due to the changes in fascicle length increasing the series compliance of the muscle fibre (Blazevich et al., 2009). Further research is required to broaden the understanding into the effect strength and power training might have on fascicle length adaptation and the consequences for dynamic performance.

Pennation angle

The pennation angle of a muscle, defined as the angle between the muscle fascicles and line of action, has an important effect on the force velocity relationship and therefore the amount of power a muscle can produce (Cormie et al., 2011b). A muscle with a greater pennation angle will be able to generate more force due to more sarcomeres being arranged in parallel which increases the likelihood that a fibre will operate closer to its optimum length (Muhl, 1982). Consequently, a greater pennation angle acts to increase a muscles cross sectional area which will influence the maximum force and power generating capabilities of a muscle. Pennation angle has been reported to increase in response to heavy strength training. In particular, heavy strength training over 14 weeks has been reported to initiate significant increases in pennation angle of the vastus lateralis which was reported to support increases in both muscle cross sectional area, volume and contractile strength capacity (Aagaard et al., 2001). In contrast, sprinting and jumping interventions have been reported to decrease pennation angle (Cormie et al., 2011b).

Motor unit recruitment

The ability of a muscle group to generate force for a given motor task is governed by the number and combination of motor units activated (Hodson-Tole et al., 2013). Motor unit recruitment has traditionally been reported to occur in a systematic order during graded voluntary contractions of increasing force, according to the 'size principle' (Cormie et al., 2011a). The size principle predicts that faster motor units (which innervate type II muscle fibres) will be recruited after slower motor units (which innervate type I fibres) have been activated, and will be the first motor units to be deactivated (Hodson-Tole and Wakeling, 2009). Whilst the size principle has been a cornerstone in the understanding of how different muscle fibres are used within a muscle, this theory predicts that slow muscle fibres will initially be used for fast contractions; a situation that is at odds with the contractile mechanisms of different fibre types. Subsequently, it has been demonstrated within human muscles of the triceps surae (during cycling at progressively faster pedal speeds), that preferential recruitment of fast fibres for fast tasks can be an activation strategy utilised for generating mechanical power when contraction velocities are high (Wakeling et al., 2006).

Certain training strategies have the potential to alter how motor units are recruited. Chronic heavy strength training has been suggested to be effective at increasing the number of activated motor units (Cormie et al., 2011a). Changes in neuromuscular activity, as characterised by an increased level of motor unit activation, have often been evidenced by elevated electromyography amplitudes following weeks to months of strength training which may reflect an increased efferent neural drive to muscle fibres (Aagaard et al., 2002, Aagaard

et al., 2000). Motor unit recruitment thresholds have also been demonstrated to shift to lower percentages of maximum voluntary contractions as a consequence of power training (VanCutsem et al., 1997). It has also been theorised, but yet to be demonstrated, that preferential recruitment of high threshold motor units may be enhanced after a period of heavy strength and power training (Cormie et al., 2011a). This training adaptation suggests that athletes are able to activate high-threshold motor units in place of low threshold-motor units to ensure the contractile properties of the muscle fibres match the mechanical demands of the contraction (Wakeling et al., 2006).

Motor unit firing frequency

The motor unit firing frequency represents the rate of neural impulses transmitted from the α-motor neuron to the muscle fibres (Cormie et al., 2011a). Muscle fibre firing frequency has a strong positive effect on the magnitude of contractile force exertion during the onset of force production (e.g. 0-200 ms) and therefore is likely to play a functional role in increasing an athlete's rate of force development (Andersen et al., 2010). Strength and power training strategies have been associated with elevated motor unit firing frequencies. For example, weightlifters display greater maximum firing frequencies during maximal voluntary contractions of the quadriceps compared with untrained individuals (Leong et al., 1999). Studies utilising electromyography analysis have also identified increases in motor unit firing frequencies in response to maximal isometric strength training of the abductor digiti minimi (Patten et al., 2001). In terms of dynamic exercise, VanCutsem et al. (1997) identified a six fold elevated incidence of doublet discharge firing (i.e. a motor unit firing two consecutive discharges in a 5 ms or less interval) of single motor units following ballistic training strategies (from 5.2% pre to 32.7% post). VanCutsem et al. (1997) concluded that dynamic strength training led to earlier motor unit activation, extra doublets and enhanced maximal firing rate, which contributed to an increase in strength and speed of muscle contraction.

Elevated discharge rates in response to training modalities involving high rates of force development have been attributed to changes in the maximal firing rates of spinal motor neurons (Aagaard et al., 2002), potentially due to reduced recurrent Renshaw cell inhibition (Wakeling et al., 2006). Subsequently, by influencing the magnitude of force and rate of force development, adaptation in motor unit firing frequency is influential in the development of muscular strength and power.

Motor unit synchronisation

Motor unit synchronisation occurs when two or more motor units are activated concurrently or more frequently to augment force production (Cormie et al., 2011a). Synchronisation may also increase the coordination and synergy of multiple muscles to promote higher rates of force

development (Semmler et al., 2002). Although limited, evidence does suggest that synchronisation is trainable, as weight trained athletes have demonstrated greater motor unit synchronisation characteristics when compared with untrained subjects (Semmler and Nordstrom, 1998). It has also been reported that synchronisation may be responsible for improved force production during complex, multi joint movements due to enhancements in the timing, amplitude and duration of muscle activity (Carroll et al., 2001b). Consequently, the plasticity of motor unit synchronisation in response to training may be an important neural adaptation that augments force transmission during sport specific tasks.

Inter-muscular coordination

Inter-muscular coordination describes the appropriate activation (both magnitude and timing) of agonist, synergist and antagonist muscles during a movement (Cormie et al., 2011a). For a given movement to direct optimal force production, agonist activation must be supported by synergist activity and decreased co-contraction of the antagonists. It is only with precise timing and level of activation and relaxation of agonists, synergists and antagonists that power flow through a kinetic chain will be optimised and performance maximised (Cormie et al., 2011a). Resistance training has successfully demonstrated increases in the maximum force expressed about a joint via improved efficiency in muscle coordination. Lower levels of knee flexor electromyogram activity during maximal isometric knee extension tasks have been attributed to reductions in antagonist muscle activation during a period of resistance training (Carroll et al., 2001a).

Adaptation in synergist muscles in response to training may also explain increases in force production observed independent of increased neural activation of agonists (Cormie et al., 2011a). Resistance training appears to induce neural alterations in coordination that are associated with learning the optimal pattern of muscle recruitment for the training task. Evidence also suggests that inter-muscular coordination adaptation strengthens excitatory neural connections between muscles that act as synergists during related functional tasks (Carroll et al., 2001b). Subsequently, multi joint strength training protocols may have the potential to improve muscle activation patterns for a large number of movements that recruit similar coordination strategies.

2.1.4 Conclusions

This section has attempted to demonstrate that strength and power are underlined by the morphological and neurological factors that dictate force production and coordination. Whilst the prescription of strength, power or a combination of the two training modalities may depend on an athlete's baseline training status (e.g. maximum force producing capability), it is clear

that both training schemes have the potential to initiate positive adaptations in the factors that improve the capacity of the central nervous system. The combination of heavy strength and power training appear to elicit substantial beneficial alterations in muscle architecture and the way the nervous system initiates the recruitment of task specific motor units. Training strategies that promote alterations in muscle cross sectional area and motor unit recruitment will therefore increase muscle agonist activation and tension capacity. The functional consequence of these adaptations may include a rise in contractile peak force and rate of force development, which may increase muscle strength and power capacities (Zebis et al., 2011).

Developing training strategies that enhance maximal force production and the rate at which force can be developed must involve consideration of the morphological factors which initiate mechanically induced muscle hypertrophy as well as the intra-muscular and inter-muscular factors that characterise neural adaptation. Due to the high capacity for flexibility of the central nervous system, it seems likely that structured strength and power training has the potential to improve the efficiency of a large number of related performance tasks due to the positive transfer effects of neurological adaptation (Figure 2.2).

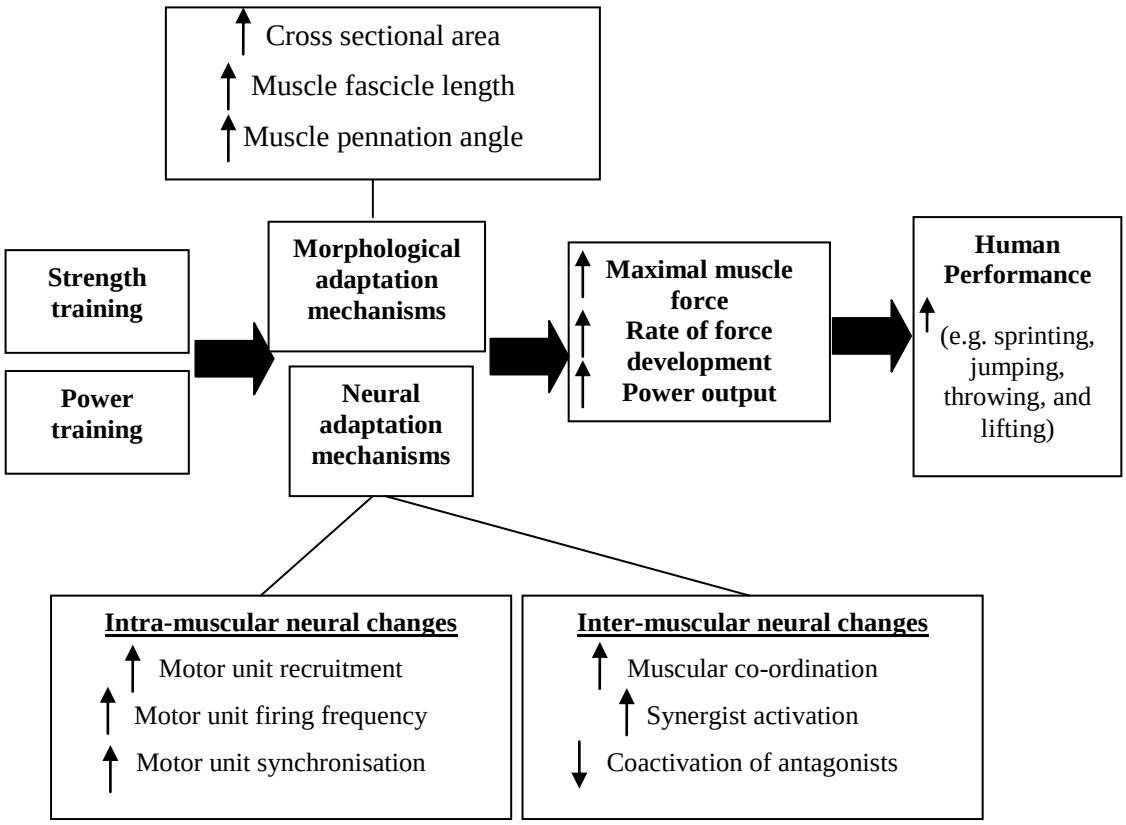

Figure 2.2 Morphological and neural changes with strength and power training.

2.2 Physiological mechanisms of post-activation potential (PAP), and the acute and repeated effects of complex training on athletic performance

2.2.1 Introduction

The contractile history of skeletal muscle has a profound effect on its ability to generate volitional force. Neuromuscular fatigue due to periods of repeated muscle activation demonstrates the negative effect contractile history may incur on muscle function. However, whilst fatigue may serve to limit force production, contractile history can also enhance subsequent contractile response, especially after high intensity muscle actions. This phenomenon is known as post-activation potential (PAP). Although fatigue and potentiation have opposing effects on force production, these two mechanisms can coexist (Rassier and Macintosh, 2000). It has been reported the force a muscle is able to generate after high intensity contractile activity is the result of the net balance between fatigue and potentiation (Docherty and Hodgson, 2007). In other words, if fatigue outweighs potentiation a decrease in neuromuscular performance will be seen, however if potentiation outweighs fatigue an increase in performance will be seen.

Studies that have successfully initiated PAP in the form of complex training have found improvements in a variety of dynamic muscular contractions. Increases in vertical jump power, peak ground reaction force, rate of force development and enhanced speed have all been identified in response to a pre-conditioning stimulus (McBride et al., 2005, Kilduff et al., 2008, Weber et al., 2008). These findings suggest complex training may be a useful tool when developing protocols aimed at maximising dynamic motor performance. The two most prevalent theories proposed to quantify the potentiated state of muscle after maximal or near maximal stimulation are: i) heightened muscle twitch force; and ii) heightened Hoffman (H-) reflex amplitude (Hodgson et al., 2005). In addition to examining the mechanisms that account for PAP, applied movement studies have also attempted to identify the various design considerations in which neuromuscular performance is maximised. It appears variables such as pre-conditioning exercise intensity (Comyns et al., 2007), recovery time (Bevan et al., 2010b), individual training status (Chiu et al., 2003) and repeated complex training exposure (Comyns et al., 2010) may all influence the effectiveness of a PAP stimulus.

The aim of this review is to provide a greater understanding into the physiological factors associated with PAP whilst also describing the programme design characteristics of complex training. This information may provide a greater insight into the functional role PAP may have as a strategy for enhancing force production. Section 2.2.2 provides a physiological overview

of the underlying central and peripheral mechanisms associated with potentiation and muscle function, whilst also reviewing the role of PAP on functional dynamic performance. Section 2.2.3 examines how manipulation of various complex training study design variables may influence the PAP effect on short-term force production.

2.2.2 PAP: Physiological overview

PAP: Underlying physiological mechanisms

Measuring muscle twitch and H-reflex response of muscle contractile properties has become a useful tool for researchers looking to study the effects of contractile history on neuromuscular response. The muscle twitch effect in particular has been proven to be a reproducible phenomenon and may form a key mechanism explaining the role of PAP in mediating peripheral changes in force production. A twitch has been defined as a brief contraction of a muscle in response to short (<1 m/s) electrical stimulation of a nerve (O'Leary et al., 1997). The force of a twitch contraction is increased following: i) a series of sub-maximal evoked twitches (MacIntosh and Willis, 2000), ii) evoked tetanic contraction (O'Leary et al., 1997), or iii) sustained maximal voluntary contractions (Gossen and Sale, 2000). These preceding forms of contractile conditioning have been shown to increase the rate of force development (RFD) in a twitch response and thus decrease its time to peak force (Hamada et al., 2000a). This effect is known as twitch potentiation (TP). Whilst high frequency electrically evoked posttetanic potentiation contraction may enhance TP, faster more forceful TP responses have been reported in response to maximum volitional muscle contraction (Gossen and Sale, 2000). It is these findings which suggest heightened TP initiated through high threshold muscle activation may influence functional motor performance.

One proposed mechanism of TP is the increase in phosphorylation of myosin regulatory light chains via myosin light chain kinase (MLCK). It has been reported that muscle stimulation which provides a TP causes an increase in sarcoplasmic calcium (Ca^{2+}) that activates MLCK (Rassier and Macintosh, 2000). Myosin light chain kinase may render the actin-myosin interaction more sensitive to Ca^{2+}, it may also be responsible for making more ATP available at the actin-myosin complex (Hodgson et al., 2005). In turn, the effects of MLCK may help increase the rate and frequency of actin-myosin cross-bridging and thus enhance force production (Docherty D, 2004). Whilst current research is limited on methods for initiating a TP via voluntary contraction, it appears that muscle fibre type and intensity of activity are key limiting factors. Hamada et al. (2000a) identified individuals who reported the greatest PAP response in the knee extensor musculature were characterised by a greater percentage of type II muscle fibre. These individuals also reported heightened TP. The TP in human tibialis anterior and plantarflexor muscles have also been found to be optimal after 10 seconds of

maximal voluntary isometric contractions: after longer contractions (with diminished intensity), the potentiation was partially suppressed by fatigue (Vandervoort et al., 1983). It has also been shown that isometric contractions <75% of maximal voluntary contraction produce little or no TP (Vandervoort et al., 1983). These findings support the role of TP as one mechanism for explaining the PAP phenomenon, as heightened neuromuscular performance in response to PAP protocols have been associated with: i) stronger athletes with greater type II fibre composition (Chiu et al., 2003), and ii) maximal isometric and/or isotonic pre-conditioning exercises greater than 85% of maximal voluntary contraction (Gullich and Schmidtbleicher, 1996, Gourgoulis et al., 2003, Young et al., 1998).

The H-reflex is another measurement tool utilised by researchers to study the effects of contractile history on neuromuscular response. The H-reflex has been defined as a monosynaptic reflex induced by an electrical stimulation of group Ia afferents of the muscle nerve (Hodgson et al., 2005). Alterations in H-reflex amplitude may influence α-motoneuron excitability and thus alter the number and activation of motor unit recruitment (Gullich and Schmidtbleicher, 1996). If H-reflex amplitude is increased post conditioning, it is assumed that in accordance with the size principle, the next motor units to be reflexly recruited would be higher threshold fast motor units. The ability to activate as many high-threshold motor units as possible will have a direct effect on the development of maximal RFD and peak force production, thus improving dynamic neuromuscular performance as a result of contractile history.

When analysing the effects of the H-reflex following volitional contraction, two main effects have been reported; i) post-activation depression (PAD); and ii) PAP. Post activation depression has been described as an inhibition of the H-reflex developing immediately upon muscle relaxation (Crone and Nielsen, 1989). The duration of PAD is dependent on the nature of the preceding contraction with research reporting PAD to be relatively short <10 seconds (Crone and Nielsen, 1989, Hultborn et al., 1996), however a PAD state has been reported up to 10 minutes post muscle relaxation (Trimble and Harp, 1998). It has been reported that depression may be due to mechanisms acting at the presynaptic level, with depression confined to the afferents activated by the pre-conditioning stimulus (Hultborn et al., 1996). The onset and duration of PAP via modulation in the H-reflex amplitude may therefore be governed by the time course of PAD

Similar to TP, heightened H-reflex of the Ia afferents can also be induced via high frequency electrical stimulation. This is also known as posttetanic potentiation, and these reflex potentiations have been reported to last anywhere from one to 16 minutes as long as stimulation frequencies are above 100 Hz (Hodgson et al., 2005). Whilst there is a large body of work examining PAD and posttetanic potentiation, only limited research studying volitional

muscle contraction and potentiation of the H-reflex exists. Trimble and Harp (1998) analysed the effect of an intense bout (eight sets of 10 repetitions) of concentric-eccentric ankle plantarflexion on the soleus and lateral gastrocnemius H-reflex response. Every subject initially displayed depression of the soleus and lateral gastrocnemius H-reflex immediately post exercise consistent with the time scale reported for PAD. After the period of depression, significant potentiation of the lateral gastrocnemius H-reflex was reported in five of the 10 subjects. This potentiation lasted up to 10 minutes post exercise. Trimble and Harp (1998) concluded that an intense bout of pre-conditioning muscle activity may ensure maximal activation of the neuromuscular system by optimising the reflex contribution to neural drive. However, it is important to note that the data reported by Trimble and Harp (1998) revealed marked inter-subject variability regarding the time course of PAD and the onset and duration of PAP.

A similar study by Gullich and Schmidtbleicher (1996) analysed the H-reflex response with subjects differentiated by their training status to assess the effect of maximal voluntary contractions of the ankle plantarflexors on H-reflex amplitude. Gullich and Schmidtbleicher (1996) found similar to Trimble and Harp (1998) that H-reflex was initially depressed for all subjects. However, following the depression H-reflex amplitude was significantly potentiated in the lateral gastrocnemius and soleus muscles only in highly trained (speed-strength) athletes occurring between four to 11 minutes. No potentiation effect was identified in the moderately trained subjects. This may be related to the greater spinal reflex processing found inherently in highly trained individuals (Hodgson et al., 2005). Together these studies suggest that the presence of a potentiated reflex response following contractile activity is dependent on an individual's strength level and the allocated recovery time before the onset of PAP. These factors may influence the effectiveness of the H-reflex contribution to motor unit recruitment and subsequent enhancement of volitional force production.

In conclusion, existing literature appears to ascribe the mechanisms associated with PAP to physiological events localised within the muscle such as the phosphorylation of myosin regulatory light chains (TP), as well as spinal level activation through increased synaptic efficacy between Ia afferent terminals and α-motorneurons (H-reflex). Currently, a limited body of research exists into the identification of these mechanisms in conjunction with measures of neuromuscular performance. Future research in which measures of dynamic force production are provided along with mechanistic analysis (i.e. TP and H-reflex) may reveal in more detail the loci of mechanisms mediating alterations in neuromuscular performance. What does seem clear is that the activation level of myogenic and neurogenic mechanisms associated with PAP are directly influenced by certain design parameters. Factors such as individual type II fibre composition and high intensity pre-conditioning exercise bouts are important for developing a TP response. Ensuring adequate recovery time post volitional muscle contraction

to allow for PAD to subside and PAP to rise as well as initiating a PAP stimulus in highly trained athletes is important for developing an effective H-reflex response. These variables may be central to whether PAP is; i) initiated via a pre-conditioning contraction; and ii) utilised to enhance motor performance.

PAP: Effects on athletic performance

Although studies examining the TP and H-reflex response in conjunction with voluntary force generation are few in number (Gossen and Sale, 2000, Gullich and Schmidtbleicher, 1996), these studies have lead researchers to apply the concept of PAP to measures of dynamic motor performance. Consequently, the short-term effects of a high intensity pre-conditioning stimulus on neuromuscular performance has been successfully identified in measures of: i) isokinetic dynometry (French et al., 2003), ii) ballistic jump performance (Kilduff et al., 2008), iii) upper body power (Kilduff et al., 2007); and iv) fast stretch shortening cycle performance (Comyns et al., 2007). Studies that have successfully incorporated the principles of PAP to enhance performance have proposed the concept of complex training as a tool to induce acute enhancements in motor performance. Complex training involves the execution of a heavy resistance exercise prior to performing a dynamic muscle activity with similar biomechanical characteristics in the belief that the heavy resistance exercise will induce a muscular potentiated state (Hodgson et al., 2005). The following paragraphs will assess current applied movement studies that have supported the role of complex training in initiating improvements in a variety of short-term performance measures.

The first study to suggest contractile history may influence dynamic muscular performance was performed by Gullich and Schmidtbleicher (1996). They found performance of four to five repetitions at 90-100% of one maximal voluntary isometric contraction (MVIC) in a single leg-press position resulted in a significant ($p < 0.01$) 3% increase in the mean of eight counter movement jumps (CMJ). Subsequent studies into the effects of a pre-conditioning MVIC have also identified improvements in dynamic performance. French et al. (2003) found performance of three sets of five second MVIC in knee extension produced significant ($p < 0.05$) improvements in a selection of dynamic performance standards which included; increased jump height (5%), maximum force qualities (5%), jump acceleration impulse (10%) and isokinetic dynometer knee extension torques (6%). French et al. (2003) concluded that performance of a maximal intensity pre-conditioning stimulus may have positive effects on subsequent muscle actions that are fulfilled in 0.25 seconds or less. The effects of a pre-conditioning stimulus on dynamic muscular contraction (<0.25 seconds) has been further supported by complex training studies looking at kinetic measures of body mass (ballistic) and barbell loaded (explosive) jump performance (Table 2.1).

The majority of complex training and acute jump squat (JS)/CMJ performance studies have utilised the back squat exercise as a potentiation stimulus at loads in excess of 86% 1RM. Force platform measurements of maximal and peak ground reaction forces (Weber et al., 2008, Duthie et al., 2002), peak RFD, peak power (Kilduff et al., 2008) and mean jump heights (Gourgoulis et al., 2003) have all been reported to be acutely increased via a complex training stimulus (Table 2.1). Jump performance with optical encoder analysis has also revealed improvements in peak and average power outputs in response to similar pre-conditioning back squatting protocols (Chiu et al., 2003, Kilduff et al., 2007). The mechanical changes associated with plyometric performance (drop jumps) in response to a high threshold back squat has also been analysed. Comyns et al. (2007) found a PAP stimulus initiated faster ground contact times, potentially due to the improved stretch shortening cycle component of the plyometric exercise via a stiffer leg spring action. Improvements in motor performance via complex training have also been reported in upper limb measures of power. Kilduff et al. (2007) reported a 5% increase in peak power output in a 40% 1RM loaded ballistic bench throw measured 12 minutes after a single set of a 3RM bench press. Gullich and Schmidtbleicher (1996) also identified significant increases in upper limb explosive strength five minutes after maximum voluntary bench press contractions.

Little research currently exists examining the effects of PAP on more functional athletic movements such as sprint performance. McBride et al. (2005) reported significant improvements (0.87%, $p < 0.05$) in 40 metre dash times in elite American football players following three repetitions of a 90% 1RM back squat. Similarly, Bevan et al. (2010b) identified significant ($p < 0.01$) improvements in 5 and 10 metre sprint times in response to a heavy pre-conditioning activity when individual responses were analysed. These studies appear to suggest that a high intensity pre-conditioning stimulus may have the potential to improve sprint performance in elite team sport athletes. However, no study to date has assessed the true effects of a pre-conditioning stimulus on acute and chronic sprint capacity in an applied sprint performance setting. Subsequently the appearance of PAP and its repeated effects on velocity-orientated performance have yet to be fully elucidated.

Results from complex training studies must also be viewed with caution as it has been reported that kinetic measures of performance display a wide variability in responsiveness to pre-conditioning muscle contractions (Chiu et al., 2003, Duthie et al., 2002, Gullich and Schmidtbleicher, 1996). Equally, the small percentage increases in kinetic performance reported in current literature is not sufficient evidence to suggest complex training will support improvements in functional athletic tasks. No study to date has evaluated the practical effectiveness of a complex training treatment in terms of the smallest worthwhile change in performance which must occur if the pre-conditioning contraction is to be considered

meaningful (e.g. effect size reporting). The lack of complex training research combining both mechanistic and kinetic analysis also limits the assumptions regarding the presence of PAP in support of enhanced performance. Performance improvements may for example be associated with a placebo effect, as previous PAP literature (Duthie et al., 2002, Kilduff et al., 2007, Kilduff et al., 2008, Weber et al., 2008) did not blind the participants to the purpose of the pre-conditioning stimulus.

Table 2.1 Summary of studies examining the short-term effects of heavy pre-conditioning contraction on subsequent dynamic jump performance.

Study	Participant number	Training status	Pre-conditioning exercise	Dependent measure(s)	Results
Chiu et al. (2003)	12 M, 12 F	7 explosive strength athletes, 17 recreationally trained athletes.	5 sets of 1 repetition back squat at 90% 1RM.	Average and peak power for loaded jumps at 30%, 50% and 70% 1RM back squat loads during RJ and CMJ.	Initially no significant effect. When divided into athletic and recreationally trained groups, significant increases in force and power measures (1-3%) identified in athletic group vs. recreationally trained (1-4% decrease).
Duthie et al. (2002)	11 F	Resistance trained (5> hours per week) team sport players.	3 sets of 3 repetition back squat at 3RM load.	Mean of 4 repetitions of loaded (30% 1RM back squat) jump squat performed for jump height, peak power and max force.	Initially no significant effect. Significant correlation between absolute max strength of participants and peak power and max force. Divided into high and low strength groups. High strength showed 2% increase in max force.
Gourgoulis et al. (2003)	20 M	Physically active men.	1 set of 2 repetition back squat at 90% 1RM.	Jump height measured from body mass CMJ performance.	Mean jump height increased by 2.9%. Participants divided into 2 groups (<160kg 1RM back squat and >160kg back squat). Stronger group showed 4% increase in

					jump height vs. 0.42% reduction in less strong group.
Kilduff et al. (2007)	23 M	Professional rugby union players.	1 set of 3 repetition back squat at 3RM load.	Peak power output from body mass CMJ performance.	Peak power increased in CMJ (8%) performed 12 minutes post pre-conditioning stimulus.
Kilduff et al. (2008)	20 M	Professional rugby union players.	3 sets of 3 repetition back squat at 87% 1RM.	Power output, jump height, and peak rate of force development measured via body weight CMJ performance.	Significant increases in peak rate of force development, power output and jump height in CMJ performance observed 8 minutes post pre-conditioning stimulus.
Weber et al. (2008)	12 M	Division 1 track and field athletes.	1 set of 5 repetition back squat at 85% 1RM.	Mean and peak jump height, mean and peak ground reaction forces from body mass JS performance.	Significant increases in peak GRF, mean jump height and peak jump height (4.6% ± 7.4%, 5.8% ± 4.8% and 4.7% ± 4.8%) observed 3 minutes after per-conditioning stimulus.

CMJ = counter-movement jump; GRF = ground reaction force; M = male; F = female; RM = repetition maximum; RJ = rebound jump.

In conclusion, evidence appears to support the short-term effects of complex training on subsequent biomechanically similar dynamic exercise. Of particular interest to athlete populations is the suggested improvement in leg spring stiffness and sprint performance in response to a high intensity pre-conditioning stimulus. This has lead researchers to suggest complex training may be a useful warm-up and or training tool for the development of dynamic motor performance. However, it has yet to be established whether the percentage changes in neuromuscular function reported in the literature in response to pre-conditioning (complex) strategies have any functional relevance to applied athletic performance. Equally, there is an absence of studies that have investigated the efficacy of complex training on long-term neuromuscular adaptation, especially when compared with other strength and power training methods.

2.2.3 PAP: Study design considerations

The magnitude and effectiveness of PAP is influenced both by the methods used to evoke it and the characteristics of the muscle. For example, an association appears to exist between type II fibre composition, muscle TP and PAP (Hamada *et al*., 2000). This may explain why certain applied movement studies have identified performance improvements only when participants were analysed based on strength status (Chiu et al., 2003, Duthie et al., 2002). It is also clear that fatigue and potentiation can co-exist (Rassier and Macintosh, 2000), and there is probably an optimal time when the muscle has recovered but is still potentiated. The following paragraphs will attempt to explore the key methodological variables which determine whether a PAP stimulus is developed and utilised for performance, or whether the PAP effect is masked by protocol design characteristics.

Pre-conditioning stimulus

The initiation of a TP in response to a pre-conditioning stimulus is a primary mechanism for the development of PAP. If a TP is to be induced via voluntary contraction then the magnitude of potentiation is dependent on both the intensity and duration of voluntary effort. A study by Vandervoort et al. (1983) was the first to identify the importance of voluntary contraction intensity on the initiation of PAP. Vandervoort et al. (1983) observed increases in TP in human tibialis anterior and plantarflexor muscles following MVIC lasting 10 seconds in duration, however when voluntary contractions were sub maximal (<75% of MVIC) little or no potentiation was observed. Maximal voluntary isometric contractions have also been associated with a heightened H-reflex response. Gullich and Schmidtbleicher (1996) found significant increases in H-reflex amplitude of the lateral gastrocnemius and soleus muscles following MVIC of the ankle plantarflexors (five repetitions of a five second MVIC) which was followed by increases in CMJ height performance. A significant improvement in jump

height, maximal force and acceleration impulse of drop jump performance has also been reported (French et al., 2003) in response to bilateral knee extension MVIC (three repetitions of a three second MVIC). It appears performing a one off MVIC of 10 seconds, or a sequence of repeated MVIC between three to five seconds, may achieve the intensity and duration thresholds required for a PAP stimulus to be developed.

Whilst currently no research has analysed the effects of weightlifting on the mechanisms associated with PAP, research indicates that weightlifting protocols are effective at initiating potentiation as long as the external loading is of a high enough magnitude. To date a number of studies have reported improvements in jump performance in response to a one off back squatting exercise. Loads of 86% 1RM for five repetitions (Weber et al., 2008, Young et al., 1998) and 90% 1RM for three repetitions (Kilduff et al., 2007, McBride et al., 2005) have all been associated with acute performance enhancement. Repeated high load back squatting protocols have also been utilised to successfully improve performance. Kilduff et al. (2008) reported improvements in kinetic measures of a CMJ in response to three sets of three repetitions back squat at loads equivalent to 87% 1RM. A study by Comyns et al. (2010) also reported significant improvements in drop jump performance only when participants performed one repetition of a back squat at a 93% 1RM load, when lighter loads (65% and 80% 1RM) were performed performance was significantly impaired. Crum et al. (2012) also reported no significant potentiation effects in measures of CMJ performance in response to a pre-conditioning back squatting protocol performed at both 50% 1RM (three sets of one repetition) and 60% 1RM loads (three sets of one repetition). Together these studies suggest that weightlifting threshold intensities above 86% 1RM is important for the benefits of PAP to be maximised. Much like maximal isometric contraction, central (H-reflex) and peripheral (TP) nervous system activation in response to a pre-conditioning weightlifting activity may be proportional to the intensity of the effort. From an applied perspective, using the heaviest load possible (e.g. 93% 1RM) for a one off effort or a repeated weightlifting protocol with loads >86% 1RM may provide an effective stimulus to initiate PAP and alter performance.

Training status

Training history and strength levels of participants seem to be important factors in the outcome of the majority of PAP studies. Studies to date have used participants of varying strength levels (from recreationally trained to elite strength and power athletes), and in some studies, it was only when participants were differentiated into “strong” and “weak” groups based on strength status that a performance effect was observed (Table 2.1). A study by Chiu et al. (2003) initially reported no change in jump performance after a pre-conditioning stimulus when the group was considered as a whole. However, once the group was divided on the basis of training status, performance increases were observed in the athletically trained group.

Similarly, Gourgoulis et al. (2003) only observed improved jump performance after a heavy back squat when the participants were split into groups based on their baseline back squat strength (strong group = >160 kg, weak group = <160 kg), with the stronger group demonstrating increases in jump performance. Duthie et al. (2002) also found significant correlations between changes in peak power and max force performance (jump squat) after a pre-conditioning stimulus and absolute measures of max strength (e.g. 1RM) (r = 0.73 and r = 0.66). Applied movement studies therefore suggest that stronger individuals are better able to utilise the PAP effect for improvements in performance compared with their weaker peers.

The association between strength and potentiation appears to be manifested in the effect on H-reflex and TP mechanisms. For example, it has been demonstrated that resistance trained athletes have significantly greater musculature activation during high intensity training (Aagaard et al., 2002), which would have a direct influence on the magnitude of H-reflex potentiation and myosin regulatory light chain phosphorylation responses. It is also possible that stronger individuals are better able to benefit from PAP because they may possess a greater proportion of type II fibres compared with their less strong peers. There is evidence to suggest that human muscle with a higher percentage of type II fibre exhibits greater PAP due to faster TP in response to evoked tetanic contractions, repetitive low frequency twitches (Sweeney et al., 1993) and MVIC (Hamada et al., 2000a). In particular, Hamada et al. (2000a) was able to identify high PAP responders versus Low PAP responders based on the participant's individual fibre distribution. Hamada et al. (2000a) found high PAP responders had a greater percentage of vastus lateralis type II fibre distribution (72 ± 9 vs. 86 ± 7 ms, P < 0.05) and shorter time to peak torque TP (61 ± 12 vs. 86 ± 7 ms, P < 0.05) than the low PAP responders. In response to the potentiation stimulus Hamada et al. (2000a) also observed an increase in the amplitude of the muscle compound action potential (M wave) in the participants with higher type II fibre percentages.

It has also been suggested that type II skeletal fibre has a greater ability to prolong and maintain the phosphorylated form of myosin regulatory light chain after brief tetanus stimulation (Sweeney et al., 1993). It appears that type II muscle fibre may be better adapted for enhancing the effects of PAP by: i) increasing the rate, force and maintenance of an induced TP and; ii) amplifying the muscle action potential (M wave) which may alter fibre membrane sodium and potassium transport mechanics and subsequently force production (Hamada et al., 2000a). In conclusion, type II fibre distribution and muscular strength status are important factors which may dictate the level to which PAP can be developed. These variables are therefore important considerations when analysing the performance effects of complex training studies

Recovery time

The duration of time allocated between the pre-conditioning stimulus and subsequent dynamic activity appears to be a key consideration when designing a complex training protocol. Conflict in the literature currently exists as to the optimal recovery time at which potentiation has its greatest effect on performance, with studies reporting times ranging from three to 18.5 minutes (Chiu et al., 2003, Kilduff et al., 2008). It is widely accepted that muscle performance following a pre-conditioning stimulus depends on the balance between fatigue and potentiation (Kilduff et al., 2007). However, the ability to enhance potentiation may be highly individualised. Trimble and Harp (1998) for example found that out of 10 participants only five were able to overcome H-reflex depression and enhance PAP, the other five demonstrated prolonged periods of PAD. Most significantly Trimble and Harp (1998) identified one to three minutes to be the transition period where depression dominated in some subjects and potentiation dominated in others.

Applied movement studies have reported four minutes to be the minimum point at which potentiation may positively influence performance in strength trained athletes. Improvements in sprint times, CMJ performance and leg spring stiffness have all been identified four minutes after a heavy back squat pre-conditioning stimulus in strength trained athletes (Comyns et al., 2007, McBride et al., 2005, Young et al., 1998). Studies that have analysed potentiation effects within four minutes have failed to identify any gains in motor performance (Crewther et al., 2011c, Jensen and Ebben, 2003). Jensen and Ebben (2003) for example found no significant effects on jump height or ground reaction forces after a pre-conditioning stimulus at 10 s, one, two, three and four minute recovery points. Furthermore, no significant effect was found when the participants were split into high and low strength groups suggesting even highly trained individuals require a period of time in which fatigue must subside and PAP develop.

The question therefore arises; what is the optimal recovery time "window" when potentiation positively influences performance? Chiu et al. (2003) found improvements in jump squat power in strength trained athletes at both five and 18.5 minute recovery points suggesting a 13.5 minute period exists in which well trained individuals may be able to utilise the effects of PAP. Chiu et al. (2003) however only looked at performance responses at these two points, subsequent research has looked at a range of time points after an initial four minutes of recovery. In particular Kilduff et al. (2007) found both CMJ and bench throw performance decreased immediately post pre-conditioning (15 seconds), was approximately level with baseline values at four minutes, significantly increased above baseline at both eight and 12 minutes and dropped back down to around baseline after 16 and 20 minutes.

In practical terms it appears the optimal time between the pre-conditioning stimulus and dynamic activity differs between athletes. Bevan et al. (2010b) for example reported eight minutes to be the time in which the majority (47%) of the study participants reported the greatest increases in sprint performance in response to a complex training protocol. Optimal increases in sprint performance were however also witnessed at four minutes for 13% of the participants, 12 minutes for 27% and 16 minutes for 13% (Bevan et al., 2010b). Similarly Crewther et al. (2011c) reported greater changes in CMJ performance as a consequence of PAP when the participants were analysed based on their individual response patterns. In conclusion, it appears that after a four minute period the performance enhancing effects of PAP may begin to be realised in stronger athletes. However the optimal time point in which maximum performance increases are obtained is highly individualised. Whilst eight to 12 minutes may be suggested based on the literature, identifying and assigning recovery periods that are optimal for the individual may provide the best practice for maximising the performance effects of PAP.

Repeated exposure

To date research has successfully identified the methodological factors which best develop PAP. However, the functional role of PAP as a training modality for the development of long-term neuromuscular adaptation remains questionable. A study by Comyns et al. (2010) analysed the repeated effects of a complex training protocol on subsequent performance. Comyns et al. (2010) exposed 11 elite rugby players to 30 metre sprints after a 3RM back squat protocol on four separate testing occasions. The session × phase interactions revealed significant improvements in the pre to post-test changes in 20 and 30 metre velocities from session one to session four. Comyns et al. (2010) concluded that repeated exposure to the PAP protocol caused progressive improvements in sprint performance and that the elite rugby participants may have learned to apply any potentiation effects. This novel finding suggests that PAP may be trainable, and that elite athlete populations may require repeated complex training exposures for the effects of PAP to successfully transfer to performance enhancement. If the potentiating effect is learnable then complex training may be an effective tool for facilitating long-term neuromuscular adaptation.

2.2.4 Conclusions

If a pre-conditioning muscular contraction is of a high enough intensity then evidence suggests that experienced athletes are better adapted at enhancing the physiological mechanisms of PAP. The greater ability of stronger athletes to excite the mechanisms of PAP may be associated with greater type II fibre distribution and spinal reflex processing. The combined effects of these adaptations may be the difference between development and utilisation of PAP

or prolonged fatigue associated with PAD. Stronger athletes are therefore better adapted at overcoming the inhibitory mechanisms of fatigue via a greater realisation of PAP during recovery periods. In practice, the development and effect of PAP through complex training depends on the identification of individualised recovery times and ensuring the performance activity has movement specificity to the pre-conditioning stimulus. However, the validity of complex training is still unclear. Future research is needed to identify whether complex training protocols: i) are effective at developing a PAP stimulus over repeated training sessions, ii) provide the progression and overload required to achieve long-term neuromuscular adaptation; and iii) provide a practical training modality in the applied field of elite athletic development.

2.3 Hormonal response to acute and chronic resistance training and the short-term effects of hormones on dynamic performance

2.3.1 Introduction

The endocrine system may play a role in strength and power development by mediating the remodelling of skeletal muscle. Specifically, biological alterations in concentrations of the anabolic hormone testosterone and catabolic hormone cortisol may mediate long-term changes in protein metabolism, muscle growth and force potential (Crewther et al., 2006). Various resistance training protocols have been shown to elicit significant acute changes in the hormonal environment (McCaulley et al., 2009a, Smilios et al., 2003). In particular, protocols high in volume, moderate to high in intensity, using short rest intervals and stressing large muscle mass tend to produce the greatest acute alterations in testosterone and cortisol (Kraemer and Ratamess, 2005). Repeated exposure to resistance training therefore has the potential to elicit chronic adaptations in the endocrine system which may promote a favourable environment for muscle growth and improvements in performance. Chronic changes in endogenous hormone balance in response to resistance training may also have increased importance for strength trained athletes, as further improvements in strength and muscle hypertrophy are much more limited than in previously untrained individuals (Ahtiainen et al., 2003). However, whilst alterations in endocrine function have been associated with changes in muscle strength and size over chronic training periods (Hakkinen et al., 1985), the exercise induced hormonal influence on muscle protein synthesis has yet to be fully quantified (West et al., 2009).

The role of testosterone and cortisol to resistance training adaptation may not be limited to long-term morphological adaptation. Exogenous changes in testosterone and cortisol concentrations have been associated with short-term (i.e. seconds, minutes to hours) improvements in central nervous system (CNS) and peripheral nervous system (PNS) mechanics. Endogenous evidence from athletic studies also provides support regarding the short-term effects of hormones on motor performance. Basal testosterone levels have been shown to predict performance in various measures of speed, strength, power and jump performance (Crewther et al., 2009b, Cardinale and Stone, 2006). These studies suggest that, for athletically trained individuals, a relationship may exist between neuromuscular performance and hormone secretion patterns. For some elite athletes, short-term changes in steroid hormones would seem one possible moderator of muscle functioning and therefore training performance irrespective of muscle growth. The acute role of testosterone and cortisol in mediating force production may therefore have implications for PAP. It may be suggested that the potentiating effects of complex training could be supported by hormonal mechanisms.

This review will look to analyse the evidence that supports the long and short-term effects of testosterone and cortisol on the neuromuscular system. This information may provide a greater insight into the potential dual role of testosterone and cortisol in mediating neuromuscular adaptation and functioning in elite athletes. Section 2.3.2 will analyse the long-term effects of hormones on muscle remodelling and performance over chronic resistance training periods, whilst identifying the varied hormonal response to training when analysed in an applied setting. Section 2.3.3 will assess the short-term effect of exogenous hormones on the central nervous system and peripheral nervous system, the endogenous hormone and performance relationship, and the implications acute hormone manipulation may have for PAP development.

2.3.2 Long-term effects of hormones

Muscle development

It is generally believed that testosterone and cortisol control long-term (genomic) changes in protein metabolism, muscle size and force potential. Testosterone is considered a primary anabolic hormone by increasing protein synthesis and decreasing protein degradation, and cortisol the primary catabolic hormone as it increases protein degradation and decreases protein synthesis (Kraemer and Ratamess, 2005). The net balance between testosterone and cortisol will influence an increase, decrease or maintenance in muscle fibre and whole muscle size (Crewther et al., 2011a). As a relationship exists between the force generating capacity and muscle cross sectional area (Bruce et al., 1997), exogenous and endogenous testosterone and cortisol concentrations may contribute to muscle performance by mediating chronic (i.e. weeks to months) changes in muscle size. Exogenous treatment of supraphysiological doses of testosterone in males has been shown to increase muscle size and strength in a dose dependent manner (Bhasin et al., 2001). Combining exogenous testosterone treatments with resistance training has also provided greater muscular adaptation than either alone (Blazevich and Giorgi, 2001). Regular training may also counteract the catabolic effects of total and free cortisol (Brillon et al., 1995) which may help promote an anabolic environment.

With the moral, legal and health issues associated with exogenous prescription of steroid hormones, mediation of the endocrine system via resistance training may be one mechanism which contributes to progressive long-term muscular development. In addition to hormonal factors, other events and pathways including; activation of satellite cells, local growth, mechanical signalling and metabolic factors (Andersen and Aagaard, 2010) as well as tendon and connective tissue remodelling (Folland and Williams, 2007), may all contribute to net changes in muscle size. This review however will focus on the hormonal contribution to long-term muscle development.

It has been reported that acute hormonal elevations associated with resistance training may increase the likelihood of receptor (e.g. peptide and or steroid) interactions on target tissue which may mediate muscle protein metabolism (Kraemer and Ratamess, 2005). Chronic resistance training may therefore initiate changes in the endocrine environment which facilitate long-term muscular adaptation. In particular, moderate intensity resistance exercise of significant volume with short rest periods appears to induce the greatest acute increases in various anabolic (e.g. testosterone, growth hormone, growth factors) and catabolic (e.g. cortisol) hormone concentrations during recovery periods (McCaulley et al., 2009a, Smilios et al., 2003). Conversely maximal strength and power workouts typically produce minimal hormonal change (Linnamo et al., 2005), so adaptive changes are generally limited to the neural pathways. With greater knowledge of the acute hormonal response to various workout designs it has been suggested that hormonal adaptations over chronic training periods may entail four general classifications:

Acute changes during and post resistance exercise;

Chronic changes in resting concentrations;

Chronic changes in the acute response to a resistance exercise stimulus;

Changes in muscle cell receptor content.

(Kraemer and Ratamess, 2005)

Whilst it appears a dose response relationship exists between extended resistance training, acute/chronic hormonal response and muscle development, the impact of repeated hormone elevations following training may vary depending on individual training experience. In particular, modifications in basal testosterone and or cortisol concentrations with resistance training appear to play a permissive role in supporting performance gains in previously untrained males. During short training periods (four weeks), negligible modifications in testosterone to cortisol ratio have been observed in non-elite athletes, whilst elite athletes identified significant increases in post training testosterone to cortisol ratio which was positively correlated to improvements in measures of power (r = 0.92) (Fry et al., 2000). Reports into longer term (10-24 weeks) strength training in untrained males have however identified progressive increases in resting and acute testosterone in conjunction with increases in muscle cross sectional area and maximum strength (Hakkinen et al., 2002b, Kraemer et al., 1999). Closer analysis of these performance and hormonal alterations however suggests the modified hormonal response occurs during later training stages (between 14 – 24 weeks), whilst performance gains occur much earlier in the training cycle irrespective of any changes in muscle size (Ahtiainen et al., 2003, Kraemer et al., 1999). These findings support the notion

that initial performance gains in untrained individuals are largely due to neural adaptations (e.g. motor unit recruitment and/or firing frequency), whilst contractile protein metabolism may predominate as training experience and endocrine adaptation develop (Hakkinen, 1989).

The modification of endogenous testosterone and cortisol concentrations in response to resistance training may however be a factor supporting muscular adaptation in well trained athletes. It has been reported that elite strength athletes have limited potential for inducing muscle growth and related performance changes through training (Ahtiainen et al., 2003, Hakkinen, 1989). Endogenous hormones might therefore play a role in neuromuscular development in this athletic population. Chronic resistance training has been shown to alter basal testosterone concentrations, reduce the overall cortisol response and increase muscle mass and strength in previously trained males (Ahtiainen et al., 2003, Hakkinen et al., 1985). The difference between trained and untrained males may be attributed to adaptive changes in the neuromuscular and endocrine systems, combined with the greater training capacity (e.g. greater capability to tolerate volume and training intensity) of trained men (Crewther et al., 2011a). It appears that whilst the training response and adaptation of endogenous testosterone and cortisol for untrained males may be permissive, a dose response role may exist for trained individuals. In particular, prolonged intensive strength training in elite athletes may initiate chronic pituitary and hypothalamic adaptations which increase resting serum testosterone concentrations (Hakkinen et al., 1988), whilst altering adrenal sensitivity and resting cortisol concentrations (Izquierdo et al., 2006).These hormonal modifications have also been used to explain accompanying changes in strength, power and speed with elite level athletes (Fry et al., 2000, Potteiger et al., 1995).

However, care must be taken when interpreting the findings of resistance training studies as elevations in circulating testosterone concentrations and increases in muscle hypertrophy and strength does not infer the existence of a cause and effect relationship. Emerging research suggests molecular responses to acute resistance exercise appear to originate from mechanical cues within the muscle cell, irrespective of circulating hormonal factors (West et al., 2010a). In particular West et al. (2010b), found that transient increases in endogenous anabolic hormones have no effect on anabolic signalling pathways or myofibrillar protein synthesis in response to an acute resistance training bout. West et al. (2010b) concluded that local mechanisms are likely to be of predominant importance for the post exercise increase in muscle protein synthesis, whilst post exercise hormonal increases cannot be used as proxy markers of skeletal muscle hypertrophic potential. It must be noted however, that this study analysed systemic hormonal concentrations and therefore no identification of the local hormonal contribution to muscle protein synthesis was made. Longer-term analysis into the molecular regulation of contractile protein remodelling via hormonal mechanisms is certainly needed, particularly in elite athlete populations

Whilst athlete training status appears to be a limiting factor influencing chronic endogenous hormonal response, other acute factors also influence the role the endocrine system plays in support of training adaptation. The different acute and chronic hormonal response to hypertrophy, strength and power workout designs have been mentioned above. Initially, it appears higher volume/moderate intensity programmes initiate the greatest long-term hormonal effect on muscle cross sectional area and performance. However, this perspective may be too simplistic given the potential for both immediate and long-term steroid effects on neuromuscular performance (e.g. maximal force and power output). It appears short-term manipulation of testosterone and cortisol have an immediate effect on neuromuscular functioning which may influence long-term adaptation in strength and power expression (Crewther et al., 2009b, Viru and Viru, 2005). The possible role of acute hormone concentrations and manipulation on muscle function and athletic performance will be discussed in section 2.3.3.

Nutritional adjustments may also offer another strategy for modifying the chronic hormonal response to training. Carbohydrate and/or amino acid supplementation has been found to be effective at acutely increasing testosterone and reducing cortisol concentrations (over 12 weeks of resistance training), which was associated with greater gains in muscle cross sectional area, muscle strength and power (Bird et al., 2006, Kraemer et al., 2009). It appears that manipulation of workout design and nutritional variables may be strategies which facilitate modifications in the hormonal milieu during resistance training, and thereafter, alter short and long-term performance outcomes.

Finally, the acute and chronic endogenous hormonal contribution to resistance exercise may be limited by genetic variation. Genetic predisposition has been reported to account for up to half of the variation in human motor performance and is therefore a major factor governing athletic success (Smith, 2003). In particular, research suggests that within homogenous athletic groups large individual differences in testosterone and cortisol concentrations are observed in response to various resistance training stimuli which may be accounted for by genetic variation (Crewther et al., 2009c, Beaven et al., 2008b). A recent study of elite male rugby players identified significant individual, protocol dependent variations in testosterone responses when the athletes were exposed to four different resistance exercise sessions (Beaven et al., 2008b) (Table 2.2). In a follow up study, Beaven et al. (2008a) suggested that resistance exercise prescription based on an individual's testosterone response may be important for promoting optimal increases in muscle cross sectional area and strength. It may be suggested that for strength trained populations, hormonal response patterns reflect genetic differences in our ability to cope with different exercise stressors (Di Luigi et al., 2003). Using hormones as genetic markers for predicting an athlete's trainability to various workouts may be a factor

worth considering when designing individual programmes aimed at maximising neuromuscular adaptation.

In conclusion, even though hormonal responses to resistance training programmes have been well characterised (Ahtiainen et al., 2003, Hakkinen et al., 1985), the influence of exercise induced hormonal regulation of training adaptation remains equivocal. Endogenous evidence from athletes suggests acute alterations in testosterone and cortisol may be associated with long-term muscular adaptation and performance. However, evidence is emerging which questions the role exercise induced hormonal responses play at initiating key intracellular signalling proteins involved in muscle protein synthesis (Atherton and Smith, 2012, West et al., 2010b, West et al., 2009). It also appears that several factors (e.g. training status, workout design, nutrition and genetics) acutely influence the testosterone and or cortisol response to training. In particular, the hormonal association to training response appears to be greater in elite athletes, possibly due to the heightened training status of the neuromuscular and endocrine systems in these athletic groups. As well as hormonal mechanisms, morphological adaptation is likely to occur as a result of several localised training factors (e.g. mechanical, metabolic) that work in concert to initiate the intramuscular signals that lead to the stimulation of muscle protein synthesis (Andersen and Aagaard, 2010). Figure 2.3 demonstrates how these training factors may integrate to help support acute and chronic muscle protein synthesis and hypertrophy in response to repeated resistance training. Long-term strength and power development, as a consequence of repeated resistance training, is therefore facilitated by a multitude of adaptive strategies.

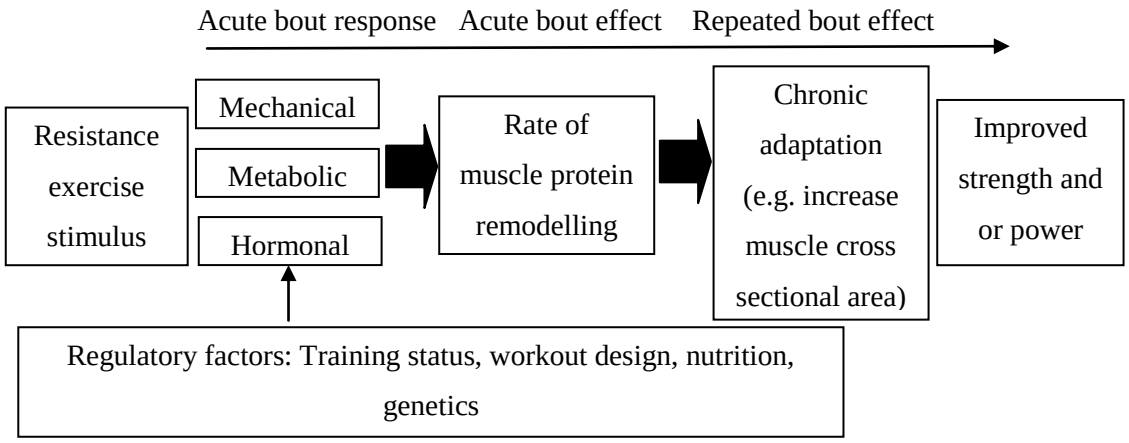

Figure 2.3 Schematic representation of the morphological pathway to adaptation via a resistance training stimulus. Adapted from Crewther et al. (2006).

Endogenous hormones and applied training

Whilst it appears resistance training studies have identified associations between neuromuscular and endocrine system development, reports into season-long performance and hormonal change in the team sport environment appears equivocal. Applied research into team sport athletes suggests the alterations in testosterone and cortisol over long-term resistance training and competitive periods demonstrate a lack of reproducibility, with little or no correlations between endocrine response and neuromuscular performance (Argus et al., 2009, Cormack et al., 2008). In particular, Cormack et al. (2008) identified progressive decreases in resting cortisol concentrations, weekly fluctuations in testosterone and a large increase in testosterone to cortisol ratio in elite Australian Rules Football players over a 22 match season. In comparison, non significant rises in cortisol, testosterone and testosterone to cortisol ratio have been observed over competitive seasons in both elite soccer (Kraemer et al., 2004) and rugby union players (Argus et al., 2009). It appears both endocrine and neuromuscular responses during in season periods appear to be highly sport specific. However, it may be suggested that competitive training and playing periods elicit fluctuations in endocrine response patterns which limit the potential for training adaptation to be supported via hormonal mechanisms (Cormack et al., 2008).

The monitoring of both hormonal and neuromuscular markers throughout a season may however provide predictive information regarding an athlete's physical readiness to train and or compete (Coutts et al., 2007). In particular, testosterone, cortisol or their ratio value could be useful in assessing the impact of training and competition as a reflection of the balance between anabolic and catabolic processes (Argus et al., 2009). The monitoring of these variables throughout a season may help identify when an individual may be at risk of neuromuscular and endocrine fatigue, and subsequently dictate when interventions such as reduction of training load or periods of rest should be implemented (Cormack et al., 2008). Further research profiling individual sports may be needed to identify the usefulness of these measures for the detection and prevention of overtraining during extended performance periods.

2.3.3 Short-term effects of hormones

Endogenous hormones and athletic performance

Support for the concept of short-term (non-genomic) hormonal effects has come from athletic studies which have identified relationships between hormone levels and performance in various tests of neuromuscular function. Cardinale and Stone (2006) reported positive correlations between testosterone levels and vertical jump performance in elite male and

female sprinters, handball, volleyball and soccer players. Similarly, Bosco et al. (1996b) found testosterone concentrations to reflect relative jumping ability in elite team sport athletes. The specific relationship between testosterone level and explosive leg strength has been highlighted by the fact that aerobic endurance, determined by a Cooper's 12-minute run test, has shown negative correlations (Table 2.3) with group basal testosterone and cortisol levels in professional soccer players (Bosco et al., 1996a). Correlations between salivary testosterone and or cortisol concentrations with various measures of speed, power and strength have also been reported in elite rugby union players (Crewther et al., 2009c). Basal testosterone has also proven to be a strong predictor of squat strength and 10 metre sprint performance in individuals with relatively high strength levels (1RM > 2.0 × body weight), but a poor predictor in less strong individuals (Crewther et al., 2012a). The correlation data for these athletic studies are presented in Table 2.3.

It appears that an individual's basal hormonal profile, more specifically testosterone level, reflects their ability to express force rapidly over a wide range of physiological effects (e.g. strength, power and speed). The temporal variance in testosterone concentrations as a predictor of performance also appears to be unique to well-trained athletic populations. Since well-developed strength athletes have limited potential for inducing muscle growth (Hakkinen, 1989), the effects of testosterone could play a greater role in the acute expression, and long-term development of the neuromuscular system in these athletic groups. The potential existence of short-term hormonal effects in elite athletes therefore provides a new paradigm for investigating athletic performance and training adaptation.

To further demonstrate the role endogenous hormone concentrations play in support of high force related activities, differences in basal hormone levels have been observed between groups with different training and competitive backgrounds. For example, elite level sprinters have reported higher total testosterone concentrations when compared with elite soccer and handball players (Cardinale and Stone, 2006) and endurance runners (Bambaeichi and Rahnama, 2005). Evidence also indicates that endurance based athletes demonstrate suppressed basal testosterone concentrations when compared to explosive event athletes such as weightlifters (Izquierdo et al., 2004). These results suggest that athletes with higher testosterone profiles demonstrate greater athletic ability in strength and or power orientated events. Individual differences in testosterone and cortisol concentrations have also been used to differentiate playing ability within the elite team sport environment. Kraemer et al. (2004) reported positive correlations between testosterone concentrations and performance changes (over the course of a season) in elite soccer players who were classified as starters. Non starters however demonstrated negative performance associations with changes in cortisol (Table 2.3). Consequently, analysing an individual's hormonal profile may offer an insight into a genetic factor that is associated with athletic performance potential both within a

homogenous group, and between sports with different physiological demands (Crewther et al., 2011a).

With the relative importance of the hormone milieu established, it may be possible to use exercise to modify hormones and performance as a form of potentiation (Hodgson et al., 2005). Repeated exercise bouts which activate an endocrine response have been shown to elicit performance improvements in subsequent efforts. Significantly higher leg power outputs have been observed in the second of two supramaximal cycle sprints which was accompanied by elevated total-testosterone levels after the first sprint (Obminski et al., 1998). Similarly, elevations in testosterone concentration in response to a 60 second repeated jump protocol demonstrated significant correlations to progressive increases in power output (r = 0.66) and jump height (r = 0.61) (Bosco et al., 1996b). Combining leg training with arm training during individual workouts has also initiated greater improvements in arm strength compared with workouts using arm exercises in isolation (Hansen et al., 2001, Ronnestad et al., 2011). The observed strength increases was attributed to the greater acute testosterone and cortisol response reported in the combined workout versus arm training alone. These results may suggest that hormonal changes induced by one muscle group could offer systemic benefits for other muscle groups. It must however be noted that Hansen et al. (2001) and Ronnestad et al. (2011) both assessed untrained individuals who may exhibit different neuromuscular and endocrine responses to well-trained athletes (Ahtiainen et al., 2003).

In practical terms, modifying the steroid hormone response via high intensity activity may have implications for complex training via an association with the performance enhancing effects of PAP. In support of this theory, Crewther et al. (2011b) initiated significant elevations in salivary testosterone concentrations via a 40 second one off cycle sprint in elite male rugby players who then, after a two minute recovery period, performed various upper and lower limb tests for strength and power. Crewther et al. (2011b) observed improved back squat (2.6 ± 1.2%) and bench press (2.8 ± 1.0%) strength when the pre-conditioning cycle sprint exercise was performed versus control (no cycle sprint), with the improvements in performance explained, in part, by changes in hormone concentrations. Direct measurement of the effects of complex training has also shown combining heavy strength and power exercise together can lead to elevations in acute testosterone response. Beaven et al., (2011) reported a favourable anabolic milieu (i.e. elevated testosterone) immediately after a complex training session which involved repeated heavy back squatting followed, after a three minute rest period, by dynamic jump squats. In contrast, the initiation of a heavy back squat protocol as a potentiation mechanism has been reported to provide an adequate stimulus for improving jump squat performance irrespective of any changes in relative hormone concentrations (Crewther et al., 2011c). The hormonal response patterns associated with different exercise formats are reported in Table 2.2.

Rapid elevations in salivary testosterone concentrations via non-physical (visual) stimuli may also influence subsequent athletic performance (Carre and Putnam, 2010, Cook and Crewther, 2012). Cook and Crewther (2012) found significant relative increases in salivary testosterone concentrations in a group of elite rugby union players in response to various video clips (erotic, humorous, aggressive and training motivational). The relative changes in testosterone correlated with subsequent improvements in back squat three repetition maximum performance across all the video sessions (mean within-individual correlation $r = 0.85$). Psychological manipulation of the pre-workout environment may therefore provide another opportunity for regulating hormonal change and subsequent athletic performance.

Table 2.2 Hormone response patterns to strength, hypertrophy, strength endurance, power and complex training exercise sessions.

Study	Athlete	Exercise session	Exercise session aim	Hormonal response (pre-post session)
Beaven et al., (2008b)	15 elite RUP	4 (sets) 10 (reps) 70% 1RM (3 mins rest)	Hypertrophy	172.3 ± 83.1 $pg.mL^{-1}$ increase in T (in 4 participants)
		3 (sets) 5 (reps) 85% 1RM (2 mins rest)	Maximum Strength	147.2 ± 47.1 $pg.mL^{-1}$ increase in T (in 5 participants)
		5 (sets) 15 (reps) 55% 1RM (1 mins rest)	Strength endurance	317.3 ± 360.3 $pg.mL^{-1}$ increase in T (in 4 participants)
		3 (sets) 5 (reps) 40% 1RM (3 mins rest)	Power	234.0 ± 113.1 $pg.mL^{-1}$ increase in T (in 2 participants)
Beaven et al., (2011)	16 semi elite RUP	3 (sets) 3 (reps) 3-RM (3 mins rest) 3 (sets) 3 (reps) 50% 1RM (3 mins rest)	Complex (strength and power)	13% increase in T
Smilios et al., (2003)	11 resistance trained men	2-6 (sets) 5 (reps) 88% 1RM (3 mins rest)	Maximum Strength	No significant increase in T C significantly decreased ($p < 0.05$) after 2, 4 and 6 sets
		2-6 (sets) 10 (reps) 75% 1RM (2 mins rest)	Hypertrophy	T significant increase ($p < 0.05$) after 4 sets C significant increase ($p < 0.05$) after 4 and 6 sets
		2-4 (sets) 15 (reps) 60% 1RM (1 mins rest)	Strength endurance	T significant increase ($p < 0.05$) after 4 sets C significant increase ($p < 0.05$) after 4 sets
Raastad et al., (2000)	9 resistance trained men	6 (sets) 3 (reps) 3-RM	Maximum Strength	17% significant increase in T ($p < 0.01$) No significant change in C
Crewther et al. (2011b)	30 elite RUP	40 s upper body cycle sprint	Power	T significant increase ($p < 0.01$) C significantly lower ($p < 0.01$)
		40 s lower body cycle sprint	Power	T significant increase ($p < 0.001$) C significantly lower ($p < 0.01$)

RUP = rugby union players, RM = repetition maximum, T = testosterone, C = cortisol

Table 2.3 Summary of the hormonal concentration-athletic performance correlations for different athletic groups.

Study	Participants number	Hormone-performance correlations
Bosco et al. (1996b)	16 SP	Tot-T vs. CMJ height (r = 0.61) & power (r = 0.66)
Bosco et al. (1996a)	32 SP	Tot-T vs. CMJ height (r = 0.43)
		Tot-T vs. 30-m sprint velocity (r = 0.47)
		Tot-T vs. Coopers test (r = -0.40)
		Tot-C vs. Coopers test (r = -0.49)
Cardinale and Stone (2006)	48 SP, HP & SR	Tot-T vs. CMJ height (r = 0.62)
Crewther et al. (2009c)	34 RUP	Sal-T vs. 10-m sprint time (r = -0.48) & 20m sprint time (r = -0.56)
		Sal-C vs. SJMP (r = 0.41)
		Sal-C vs. BS (r = 0.39)
		Sal-T/C ratio vs. BTPP (r = 0.41)
Crewther et al. (2012a)	10 RUP	*Strong Group*
		Free-T vs. BS (r = 0.02)

		Free-T vs. 10-m sprint time (*r* = -0.87) *Weaker Group* Free-T vs. BS (*r* = 0.35) Free-T vs. 10-m sprint time (*r* = -0.18)
Kraemer et al. (2004)	25 SP	*Starters* Tot-T vs. MIKF (*r* = 0.55 to 0.61) Tot-T vs. MIF (*r* = 0.64 to 0.71) Tot-T/C ratio vs. CMJ height (*r* = 0.65) *Non Starters* Tot-T vs. CMJ height (*r* = 0.77) Tot-C vs. CMJ height (*r* = -0.64 to -0.59) Tot-C vs. 20 yard sprint times (*r* = -0.78 to -0.57) Tot-C vs. MIKF (*r* = -0.56)

Tot-T = total testosterone, Tot-C = total cortisol, Tot-T/C ratio = total testosterone to cortisol ratio, Free-T = free testosterone, Free-C = free cortisol, Sal-T = salivary testosterone, Sal-C = salivary cortisol, Sal-T/C ratio = salivary testosterone to cortisol ratio, SP = soccer players, HP = handball players, SR = sprint runners, RUP = rugby union players, CMJ = counter movement jump, MIF = maximal isometric force, MIKF = maximal isokinetic force, BTPP = bench throw peak power, BS = back squat, SJMP = squat jump mean power.

It appears that intense leg exercise via cycle sprint (Crewther et al., 2011b) or weightlifting protocols (Hansen et al., 2001), or the use of visual stimuli (Cook and Crewther, 2012), may all be effective at initiating elevated hormonal responses. However, ambiguity exists as to the optimal intervention for modifying hormonal concentrations. The fact that hormonal responses to different physiological and psychological stimuli may be highly individualised, even within homogenous athletic groups (Beaven et al., 2008b, Cook and Crewther, 2012), makes recommendations speculative. However, although limited, evidence is emerging which suggests warm-up strategies (physical and or visual) with the purpose of acutely elevating hormones, could provide a novel method for improving workout performance (e.g. greater workout strength and or power expression). Further work is needed to identify various strategies which modify hormonal response patterns as a method of performance potentiation during acute training sessions, and the long-term adaptive effects on the neuromuscular system.

In conclusion, acute basal hormonal concentrations appear to demonstrate strong correlations to measures of dynamic activity in elite athlete populations. This suggests the hormonal milieu, in particular testosterone, has an immediate effect on neuromuscular functioning and motor performance. Hormonal analysis may potentially have two practical implications for athletic performance. Firstly, monitoring basal testosterone and or cortisol concentrations within a sport could provide useful information regarding athlete performance potential and subsequently talent identification and selection (Macarthur and North, 2005). Secondly, the inclusion of warm-up strategies (physical and or visual), which modify the acute hormonal environment may be useful for potentiating training performance, particularly in elite strength athletes. If these acute effects are repeated, then the cumulative response over time may be a heightened training effect. With morphological adaptation difficult to achieve in strong athletes (Hakkinen, 1989), programmes geared at improving strength in these groups require more sophisticated training designs (Cormie et al., 2010b). Utilising the short-term actions of hormones may therefore provide a novel stimulus for initiating training variation in order to maximise training outcomes in advanced athletic groups. These conclusions must be considered with caution however, as short-term hormonal effects have yet to be consistently established as an effective training and monitoring tool in an applied environment.

Exogenous hormones and neuromuscular function

Endogenous evidence from athletic studies suggests a cause and effect relationship exists between steroid hormones and performance. If short-term (non-genomic) hormonal effects help to regulate performance capacity, then it is important to understand the physiological interactions between hormones and the neuromuscular system. The neuromuscular system controls human movement by the collective interaction between the central nervous system

(CNS) and the peripheral nervous system (PNS). Briefly, the CNS generates the signals which tell the PNS to activate and co-ordinate muscle force for the expression of human movement and athletic tasks (Crewther et al., 2011a). It has been suggested that steroid hormones have the capacity to exert different physiological effects on both the CNS (i.e. brain, spinal cord) and the PNS (i.e. motor unit-neuron and all the fibres it innervates (Crewther et al., 2011a). These effects are believed to be mediated via steroid interactions with specific membrane receptors such as; ion channels, neurotransmitters, protein kinases and G-proteins (Falkenstein et al., 2000). This section will examine the exogenous non-genomic effects of testosterone and cortisol on both the CNS and PNS.

Central nervous system

Hormones rapidly influence neuronal activity of the CNS which explains why steroid hormones have been associated with changes in mood, behaviour and cognitive function. For example, exogenous treatments of testosterone and cortisol in animal studies have been associated with alterations in measures of anxiety, memory and reproductive behaviour (Aikey et al., 2002, James and Nyby, 2002, Lacreuse et al., 2009). In vitro hormonal application has therefore demonstrated a regulative role exists for testosterone and cortisol in neuronal excitation (Smith et al., 2002) and inhibition (Zaki and Barrett-Jolley, 2002) within different regions of the animal brain. These responses are thought to be mediated by steroid interactions with non-genomic membrane bound receptors (e.g. androgen, estrogen, glucocorticoid and neurotransmitter) and ion channels (Falkenstein et al., 2000). Subsequently, testosterone and cortisol may act as "neuroactive steroids" due to their association with CNS modulation of brain function (Aloisi and Bonifazi, 2006). Although research on humans is limited, support for a steroidal role in regulating cognitive function does exist. Exogenous steroid administration in humans has been associated with fear reduction (Hermans et al., 2006), decision making (Putman et al., 2010) and memory capabilities (Buss et al., 2004). Similarly, disturbances in behaviour, mood as well as increments in aggression and depression have all been reported in athletes who have used androgenic-anabolic steroids over short and long-term periods (Hartgens and Kuipers, 2004).

As well as steroid effects on brain activity and behaviour, the motor system may provide another steroid target. Hormones may regulate CNS transduction of spinal pathways directly involved in skeletal muscle activity. For example, corticospinal motorneuron thresholds have been associated with testosterone concentrations in humans (Bonifazi et al., 2004). In this study, Bonifazi et al. (2004) increased the gonadal steroid concentration of six healthy males via a single intramuscular injection of human chronionic gonadotropin. Subsequently, all subjects demonstrated a significant decrease in the cortical motor threshold concomitant with a significant increase in plasma testosterone and oestradiol plasma concentrations (Bonifazi et

al., 2004). In comparison, the plasticity of the human motor cortex appears to be associated with circadian changes in endogenous cortisol concentrations. Sale et al. (2008) initiated electrical stimulation of the motor cortex in 25 subjects and found the motor evoked potential of muscle was significantly increased in the evening (when endogenous cortisol levels are low) but not in the morning (when endogenous cortisol levels are high). It appears that both testosterone and cortisol demonstrate an excitatory or inhibitory effect on human motor cortex potentiation. Together, the hormonal data presented have possible implications for transmitting and processing neural information to and from skeletal muscle.

Peripheral nervous system

The PNS has also been shown to be responsive to the effects of steroid hormones, with exogenous analysis suggesting testosterone and cortisol may affect synaptic transmission at the neuromuscular junction (Blanco et al., 2001). In vitro administration of cortisol on rat diaphragm muscle found that low cortisol doses potentiated action potentials and increased resting membrane polarisation due to cortisol effect on membrane permeability and changes in ion channels (Dlouha and Vyskocil, 1979). Similarly, chronic exogenous testosterone treatment on rat diaphragm muscle demonstrated a direct effect on neuromuscular transmission which was associated with reduced peripheral muscle fatigue (Blanco et al., 2001). Of particular interest was that the observed reduction in muscle fatigue was most pronounced in type IIx fibres. Research into humans has also identified an association with hormonal effects on muscle fatigue mechanisms, particularly in fast-twitch muscle fibres. Bosco et al. (2000) reported a strong correlation between changes in endogenous testosterone concentration and electromyography/power ratio (r = 0.90) for trained athletes in response to a single heavy resistance protocol. Bosco et al. (2000) suggested that this large association was an indication of testosterone influence on fast-twitch muscle fibre activity. Consequently, the authors proposed that testosterone might compensate for fatigue by improving neuromuscular efficiency in type II muscle fibres. Together, these results suggest testosterone and cortisol may contribute to fatigue regulation through mechanisms associated with PNS transmission. These effects may be particularly prevalent in type II motor units and muscle fibres.

Increasing evidence also suggests exogenous treatments of steroid hormones may initiate the rapid activation of intracellular secondary messengers important for muscle contraction (Anttila et al., 2008, Vicencio et al., 2011). In particular, animal cell model research has demonstrated fast (one to two minute) transient increases in intracellular calcium (Ca^{2+}) in response to testosterone exposures (Estrada et al., 2003, Estrada et al., 2000). Consequently, acute testosterone activated increases in Ca^{2+} may represent an intermediate step in the downstream activation of actin-myosin cross bridge formation. More recently, animal studies have demonstrated acute steroidal effects on muscle twitch potentiation (TP) and force

production, particularly in fast twitch muscle fibre (Hamdi and Mutungi, 2010a, Nguyen et al., 2005). A study by Hamdi and Mutungi (2010a) demonstrated that exogenous treatments of dehydrotestosterone (DHT) on isolated mammalian skeletal muscle lead to elevations in the phosphorylation of myosin regulatory light chain. It was postulated that the phopshorylation of MRLC potentiated the muscle twitch response by increasing the sensitivity of the contractile apparatus to Ca^{2+}, and thus increasing the potential for actin-myosin cross bridge cycling rate (Hamdi and Mutungi, 2010a). Taken together, intracellular analysis suggests that steroid administration may influence a non-genomic effect on muscle force potential via the dual response of Ca^{2+} influx and a heightened phosphorylation of the mechanisms initiating muscle contraction. These acute intracellular effects also demonstrate an association between steroid responses and PAP, and again like PAP, appears to be most pronounced in fast twitch muscle fibre.

Besides the phosphorylation of myosin regulatory light chain, testosterone and DHT administration has also demonstrated an acute signalling effect on the mitogen activated protein kinase/extracellular signal regulated kinase (ERK)-1 and ERK-2 pathways responsible for the transcriptional regulation of genes important for cell proliferation and differentiation (Estrada et al., 2003, Hamdi and Mutungi, 2010a, Nguyen et al., 2005). Therefore, it is likely that in addition to the mediation on force potentiation, acute steroid effects on downstream protein regulation may act as a point of cross-talk between non-genomic and genomic steroid signalling (Estrada et al., 2003, Hamdi and Mutungi, 2010a, Vicencio et al., 2011).

It is also believed that both testosterone and cortisol can have a direct effect on skeletal muscle energy metabolism. In particular, cortisol has been reported to increase metabolism of glycogen, free amino acids and lipids via the stimulation of glycogenolysis and gluconeogenolysis pathways (Viru and Viru, 2004). A study by Brillon et al. (1995) demonstrated that high doses of exogenous hydrocortisone treatments (80 and 200 micrograms.$Kg^{-1}.h^{-1}$) in humans lead to a significant increase in free fatty acid and amino acid availability and oxidation, which in turn, lead to a significant rise (9-15%) in resting metabolic rate. Animal model studies have also demonstrated testosterone to have a regulatory role in metabolic substrate kinetics. Ramamani et al. (1999) demonstrated that testosterone treatments in rat skeletal muscle lead to increases in adenosine triphosphate (ATP) production via an up-regulation in anaerobic enzyme activity of the phosphagen system (e.g. creatine phosphokinase and myokinase). Therefore, the metabolic effects of testosterone may be associated with high intensity performance when a reliance on anaerobic ATP production predominates.

2.3.4 Conclusions

Whilst it is traditionally believed that endogenous testosterone and cortisol help regulate long-term intracellular responses associated with muscle growth, particularly with resistance exercise, recent molecular analysis has cast doubt over this genomic theory (Atherton and Smith, 2012, West et al., 2009). Consequently, some authors suggest a lack of evidence exists demonstrating a significant physiological hormonal contribution to training induced hypertrophy and strength (West et al., 2010b). The belief that hormonal effects under post-exercise conditions are dissociated with muscle protein synthesis may however be a too simplistic perspective. It is more likely that evoked alterations in the anabolic hormonal environment may be one of many important and highly individualised proximal signals which work in concert to regulate skeletal muscle hypertrophy. Further training intervention and mechanistic analysis is required to identify the extent to which hormones initiate acute and chronic morphological adaptation in athlete populations. Physiological evidence confirms however that testosterone and cortisol may be important for the short-term (non-genomic) mediation of neuromuscular performance and training adaptation. Figure 2.4 demonstrates the potential role short and long-term hormonal effects may have on human motor function and training adaptation.

It is believed that these short-term hormonal mechanisms involve immediate potentiation of the CNS and PNS, and appear to exert a greater effect in type II motor units and fibres. Consequently, an elevated anabolic profile may support the heightened neuromuscular responses associated with PAP (e.g. steroidal effect on twitch potentiation via myosin regulatory light chain phosphorylation). Modification of the hormonal environment may therefore provide an effective stimulus for initiating an elevated training response (i.e. greater workout strength or power expression), particularly in elite athletes. However, further research is needed which demonstrates these short-term hormonal effects under physiological conditions (endogenous hormones) rather than experimental conditions (exogenous hormones), particularly within the applied athlete setting. Finally, it appears that hormonal response patterns to various stressors may be genetically governed (Beaven et al., 2008b) and therefore highly individualised. This makes recommendations regarding optimal strategies (e.g. exercise, nutritional, psychological) for manipulating endogenous hormones difficult to identify, even within homogenous groups. The short-term effects of hormones on the expression and development of the neuromuscular system for elite athletes, and the potential association with PAP over acute and chronic training periods requires further examination.

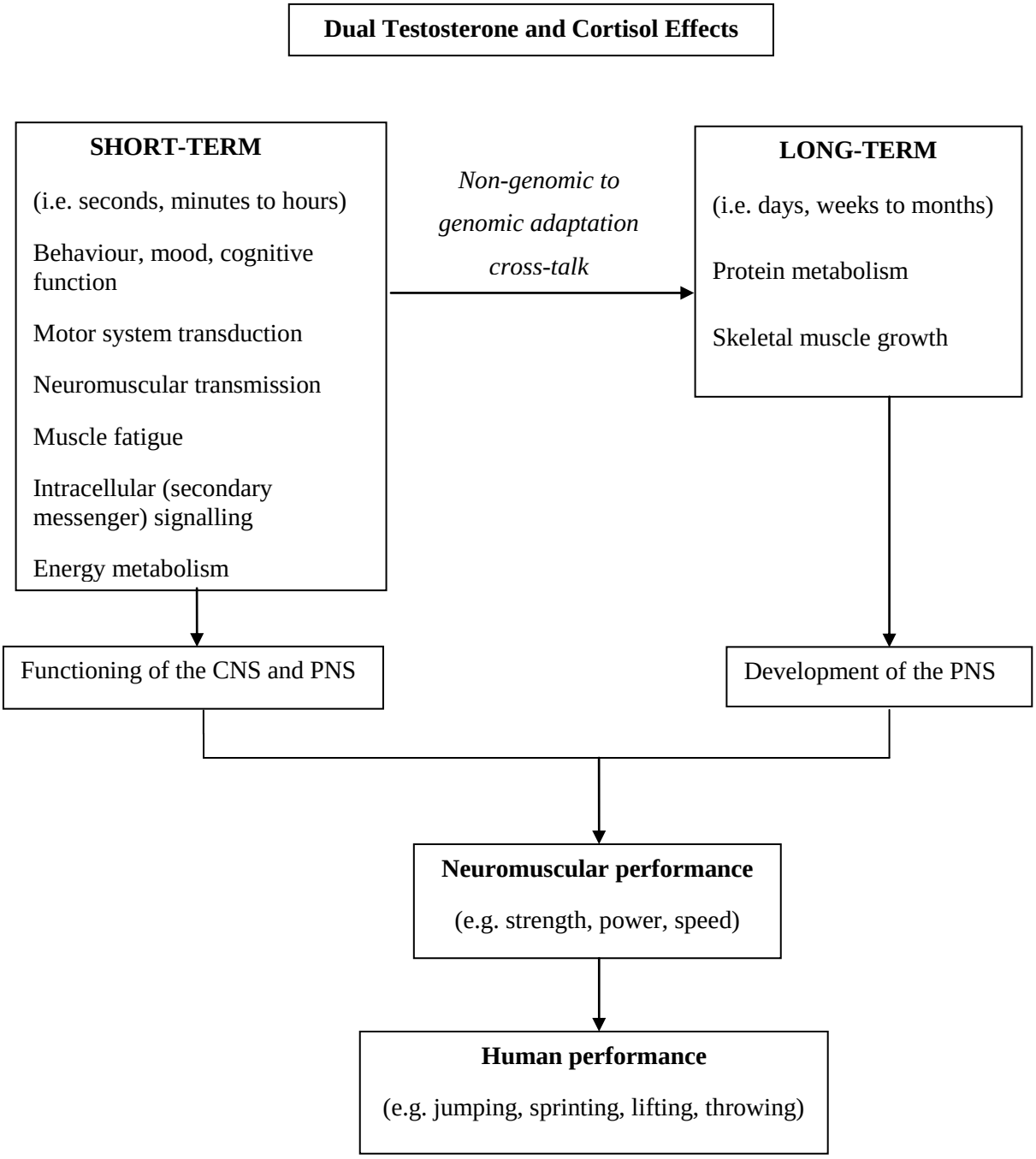

Figure 2.4 Schematic representing the potential short and long-term T and C effects on the neuromuscular system and human motor performance. Adapted from Crewther et al. (2011a).

2.4 The role of high-intensity interval training (HIT) in elite team sport athletes

2.4.1 Introduction

High-intensity interval training (HIT) has been reported to be an effective means of improving the endurance performance of elite athletes through positive adaptations in cardiovascular and skeletal muscle metabolic function. High-intensity interval exercise is characterised by brief intermittent periods of muscle contraction performed at an intensity close to that which elicits peak oxygen uptake (Gibala, 2009). It is believed that accumulating several minutes of exercise (per session) at intensities greater than 90% of VO_{2max} is required to stimulate cardiopulmonary and peripheral muscle adaptations (Buchheit and Laursen, 2013b). Whilst HIT may be performed with long work intervals (1 to 4 minutes), the use of shorter work intervals (10-60 s), repeated sprints (3-7 s) and all-out efforts (30 s), interspersed with passive to low intensity recovery has become widely utilised in applied sport environments (Buchheit, 2014). Consequently HIT exercise requires an athlete to recover and reproduce high power outputs repeatedly. Short HIT exercise strategies may therefore represent a specific and time efficient training mechanism for developing the adaptive pathways required to improve aerobic performance in elite intermittent team sport athletes.

Whilst HIT is infinitely variable with specific adaptations determined by factors related to the precise nature of the exercise stimulus, the present review will analyse the effects of low volume "short" HIT (work periods <30 s), as this form of training is most commonly used in athletic settings. The aim of this chapter is to provide an insight into the physiological and performance adaptations associated with HIT and to highlight the programme design characteristics which may influence the effectiveness of HIT strategies in an applied training environment. Section 2.4.2 will assess the influence of HIT on improving repeated exercise capacity via adaptations primarily in the oxidative capacity of skeletal muscle. Section 2.4.3 identifies some of the practical considerations for developing HIT strategies that facilitate the accumulation of high intensity exercise via appropriate manipulation of training design factors.

2.4.2 HIT: Athletic performance and physiological responses

To date HIT has repeatedly demonstrated its proficiency as a resourceful training modality for improving exercise capacity in trained athletes (Laursen and Jenkins, 2002, Lindsay et al., 1996). The use of HIT over repeated exposures has successfully demonstrated improvements in performance markers associated with both the capacity (i.e. peak power) and repeatability (i.e. endurance potential) of muscle contraction (Hazell et al., 2010). To demonstrate this, improvements in performance measures associated with aerobic power such as exercise time to

exhaustion, time trial performance as well as anaerobic power measures such as repeated sprint ability, maximum and mean power outputs have all been reported to occur in athletic groups in response to regular HIT (Gibala et al., 2012, Jacobs et al., 2013, Burgomaster et al., 2005). The adaptive performance potential of HIT may also be achieved with significantly lower weekly training volume (~90% lower in HIT) and time commitment (~67% lower in HIT) in comparison to traditional endurance training (i.e. 40-60 min cycling at 65% VO_{2max}) (Gibala et al., 2012). The practicalities and adaptive potential of HIT ensures this mode of training is regularly utilised in time limited concurrent team sport training environments. Consequently, recommendations such as six sessions of HIT, totalling approximately 15 minutes of total work, accumulated over two weeks of training have been proposed to be sufficient to stimulate the adaptive responses required to improve endurance exercise in elite athletes (Burgomaster et al., 2005).

The response pathways initiated through HIT appear to be driven primarily by changes in peripheral (skeletal muscle) and to some degree central (cardiovascular) metabolic function. In terms of the cardiovascular system, short HIT has been associated with improved oxygen carrying capacity through enhancements in cardiac output. As HIT requires training at an intensity that facilitates peak stroke volume (both during the exercise and possibly in recovery), the accumulation of repeated intervals may be sufficient to support adaptations in stroke volume capacity (Lepretre et al., 2004). It has also been demonstrated that two weeks of Wingate based HIT (30 s "all out" cycling efforts against a supra-maximal workload) and maximal sprint interval training, increases peak oxygen uptake (V_{o2peak}) (Gibala et al., 2012, Macpherson and Weston, 2014). Reduced recovery intervals programmed alongside repeated all-out 30s and 10s efforts (i.e. 2-4 minutes of recovery) has also been shown to illicit significant increases in VO_{2max} (9.2-9.3%), with the improved aerobic adaptation due to the incomplete recoveries forcing muscle metabolic pathways to regenerate energy at high rates with a decreasing anaerobic contribution (Barnett et al., 2004, Hazell et al., 2010). Even shorter work duration HIT (e.g. 10, 15 and 20s all-out efforts) have been shown to provide an effective stimulus to initiate significant increases in V_{o2peak} (15-20%) in sedentary but healthy men and women respectively (Metcalfe et al., 2012). These studies demonstrate that reduced total exercise duration and elevated sprint interval intensity can provide significant improvements in the centralised aerobic factors which govern cardiovascular performance. However, enhanced VO_{2max} in athletic groups through HIT is not a universal outcome (Jacobs et al., 2013). Consequently, cardiovascular responses to HIT could be fitness dependent, with fitter athletes less likely to benefit from this type of physiological adaptation (Buchheit and Laursen, 2013b).

In contrast, the molecular mechanisms underlying skeletal muscle metabolic adaptations to HIT appear to represent robust training outcomes that support performance enhancements for

athletic groups. Muscle peripheral responses such as increased muscle content of proteins associated with the transport and oxidation of glucose and fatty acids, and reduced non-oxidative energy provision during exercise, have all been documented after several weeks of short HIT (Burgomaster et al., 2008, Laursen, 2010). In particular, it has been reported that repeated all out 30s HIT protocols produce similar changes in skeletal muscle markers of carbohydrate metabolism to aerobic training (Burgomaster et al., 2008). HIT strategies utilising short all-out efforts have reported significant improvements in the glycemic control (i.e. insulin sensitivity) in sedentary young adults which has been partly associated with an up regulation in the glucose transporter protein GLUT4 (Babraj et al., 2009). Glucose transport has been reported to be a rate limiting step in glucose disposal (Houmard et al., 1991), therefore training induced elevations in GLUT4 may be an important adaptation governing muscle glycogen metabolism during contractions performed under conditions of high metabolic stress (Gillen et al., 2014)

Increasing the oxidative capacity of skeletal muscle through the initiation of mitochondrial biogenesis also appears to be a key outcome associated with HIT (Little et al., 2011). Mitochondrial biogenesis is characterised by increases in mitochondrial density and oxidative enzyme activity, which can improve skeletal muscle fatigue resistance and endurance performance through enhanced oxygen extraction and utilisation capacity (Hawley, 2009, Jacobs et al., 2013). Consequently, studies have tracked the activation of peroxisome proliferator activated receptor gamma coactivator (PGC-1α), which is regarded as the "master regulator" of mitochondrial biogenesis as a means of quantifying the adaptive efficacy of HIT.

In particular, Wingate based HIT has been shown to increase PGC-1α several fold when measured three hours post exercise due to increased mRNA expression of several mitochondrial genes including activated protein kinases (AMPK) and the mitogen activated protein kinases (MAPK) (Little et al., 2011). Elevated levels (approximately 100%) of skeletal muscle PGC-1α protein content has also been observed following six weeks of repeated bout short HIT in young healthy individuals (Burgomaster et al., 2008). Six weeks of short HIT (3 × 20s all-out cycle sprints interspersed with 2 minutes of recovery), performed three times each week in healthy men and women, has resulted in significant increases in maximal activity of citrate synthase which is reported to be a key indicator of mitochondrial content in human skeletal muscle (Gillen et al., 2014)). Whilst it appears that repeated dosages of low volume high intensity exercise has the capacity to trigger an intracellular signalling cascade that mediates mitochondrial protein synthesis, the minimum total volume of HIT required to maximise this response over repeated training exposures has yet to be identified. In particular, no evidence to date exists which tracks the early and late time course of mitochondrial adaptation to HIT in well trained athletes. This area of research would be useful for exercise

practitioners looking to prescribe the most effective dosages (i.e. intensity and volume load) of HIT to maximise the oxidative adaptation potential of skeletal muscle in elite athletic groups. Nevertheless, evidence appears to suggest that the mitochondrial biogenesis pathway provides the key adaptive potential for improving the peripheral aerobic power and endurance capacity of skeletal muscle.

The positive effects of central and peripheral adaptations demonstrated to occur in response to the various forms of short HIT will have substantial implications for improving performance in team sports such as rugby union. Rugby union includes aspects of prolonged submaximal exercise at lower movement velocities punctuated by bursts of high intensity efforts at high movement velocity or under contact situations (Roberts et al., 2008). Improving the central components of aerobic capacity (e.g. VO_{2max}) will therefore allow athletes to operate at a lower relative intensity during the physiological aspects of the game when ball in play time is high but intensity of effort is low. Subsequently, the peripheral adaptations associated with HIT (e.g. mitochondrial biogenesis) will improve fatigue resistance due to enhanced recovery potential during the aspects of the game when metabolic contraction intensity is high and the relief interval durations are incomplete.

2.4.3 HIT: Programme design considerations

Proper manipulation of programme design variables is essential to optimise post exercise adaptation to HIT (Buchheit and Laursen, 2013b). Positive adaptations in skeletal muscle function (e.g. mitochondrial biogenesis) have been attributed largely to the volume of work accumulated at high exercise intensity (Edgett et al., 2013). High exercise intensity also requires elevated force production, which may initiate greater neuromuscular recruitment and adaptation potential in a wider spectrum of higher threshold motor units (i.e. type II muscle fibre) (Buchheit and Laursen, 2013b, Schoenfeld et al., 2014). Ensuring HIT interventions are programmed with the correct blend of exercise volume and intensity is therefore of critical importance if improvements in the oxidative potential of skeletal muscle fibre is to be achieved. In particular, factors such as the duration of work and relief interval, the number of intervals performed, and the exercise modality employed, can all be manipulated to influence the level of metabolic and neuromuscular engagement of a given HIT protocol.

A greater quantity of research has been published over recent years describing the impact that sprint interval duration, number and intensity may have on the potential to initiate the central and peripheral adaptation mechanisms associated with HIT. A large focus on Wingate based HIT programming (4-6 × 30s all-out efforts separated by 4 minutes recovery) has clearly demonstrated its effectiveness to achieve similar levels of improved skeletal muscle oxidative capacity in shorter time frames with reduced training volume when compared to traditional

longer duration endurance training (Gibala et al., 2012, Burgomaster et al., 2008). However, evidence is now emerging to demomnstrate similar levels of positive adaptation can be achieved when the sprint numebr and duration is reduced further (i.e. 10-20s inetrvals), which has even more practcial application for athletes requiring time efficient HIT protocols. Hazell et al. (2010) compared aerobic adaptive training outcomes between HIT protocols comprised of 4-6 bouts over a two week period of; i) 30 s work duration followed by 4 minutes recovery, ii) 10 seconds work duration followed by 4 minutes recovery, and iii) 10 seconds work duration followed by 2 minutes recovery. Hazell et al. (2010) demonstrated comparable significant improvements in aerobic measures of performance between both 10 second protocols and the 30 second protocol potnaitailly indicating it is the intensity initiated during the first few seconds of each spirnt inetrval (i.e. peak power output) rather than the total work completed which is reesponsible for HIT adaptation.

More recently lower total volumes (i.e. number of repetitions) of short sprint intervals have also provided significant improvements in aerobic function. Metcalfe et al. (2012) demonstrated positive adaptation to HIT via a protocol consisting of 10-20 second all out efforts performed once in the first week of training followed by two all-out bouts per session in weeks three to six. Similalry, a protocol requiring only three minutes of intense work per week (3 × 20s all-out sprint intervals, 2 minute recovery, 3 times per week) has been proven to be of sufficient volume to improve skeletal muscle markers of oxidative capacity (Gillen et al., 2014). It can be stated therefore that short work periods of 10-20 seconds appear to be valid for prescribing work interval durations when designing HIT formats. It may be hypotheised that it is the greater relative intensity of effort, due to the continued attempt to generate maximal power during repeated shorter sprints, that stimulates the physiological stress by which adaptation is initiated (Hazell et al., 2010). These effects appear also to be achieved with dramatically reduced total volume (i.e. 1-3 bouts per session).

However, the effects of significantly reduced session volume and elevated work intensity has yet to be validated in individuals who possess higher baseline levels of aerobic fitness. It is not known for example whether the low trainig volumes recently reported in sedentary or untrained participants (e.g. 1-3 bouts per HIT session), is sufficient to initiate the central and peripheral mechansisms associated with HIT adaptation in elite athlete populations. In a recent review by Buchheit and Laursen (2013b), it was reported that the number of intervals performed should be designed to ensure the total exercise time per session exceeds four minutes of all-out effort for elite athlete groups. For example, 20 repetitions of 15 seconds all-out sprint interval durations separated by 30 second relief intervals might enable an athlete to spend approximately 5 minutes at an adaptive training intensity. Further research which analyses the optimal programme prescription and dosage for performance enhancement through short HIT in elite athletes is required.

As well as prescribing HIT intensity via the administration of all-out efforts (i.e. maximal effort each sprint), HIT can also be administered with reference to the exercise velocity or power which is associated with an athlete's VO_{2max} (i.e. maximal aerobic speed or maximal aerobic power). These intensity characteristics are commonly estimated through incremental ramp tests to exhaustion (running or cycling). Once an appropriate velocity or power output (which facilitates a sustained training intensity) is identified, appropriate work interval durations can then be prescribed. As short sprint time durations of 10-20s may provide sufficient work periods for the generation of peak power and therefore high training intensities,, coaches in sporting environments generally administer multiple efforts with work to relief ratios between 1:1-3 (i.e. relief interval durations of between 10-60 seconds) (Buchheit and Laursen, 2013b).These HIT configurations are reported to allow athletes to accumulate exercise time at high VO_2 responses during the work intervals (e.g. approximately 110-120% VO_{2max}), whilst elevating anaerobic glycolytic energy requirements (Buchheit and Laursen, 2013b).

The level of exercise intensity achieved during a given HIT protocol may also differ depending on the exercise modality used. For example, cardiorespiratory load and neuromuscular engagement of a HIT protocol can vary when running is performed with or without the inclusion of changes of direction (Buchheit, 2014). Since most intermittent team sports require frequent changes of direction, inclusion of this movement capacity may increase training specificity. However, administration of training targets must also consider the effect of change of direction to ensure total cardiovascular stress is not decreased due to the increased time required to perform effective changes of direction movements (Buchheit, 2014). Consideration must also be made regarding the effects of other HIT formats. In comparison to running based HIT, short cycle HIT may modulate variations in the physiological strain developed during exercise. In particular, differences in anaerobic glycolytic energy contribution and neuromuscular load may be observed during cycling when compared with straight line running (Buchheit and Laursen, 2013a). It is also important to consider the holistic training effects when choosing HIT formats as exercise mode (running, cycling), ground surface (synthetic track, grass, treadmill) and terrain (uphill, downhill) are all factors which may have direct implications on traumatic and overuse injury risk (Buchheit and Laursen, 2013a).

2.4.4 Conclusions

It is clear HIT has the potential to improve aerobic performance via increasing an athlete's capacity to transport, extract and utilise oxygen more effectively during repeated high intensity fatiguing muscle contractions. In order for HIT to stimulate the adaptive process' which improve cardiovascular and muscle metabolic capacity, training sessions must be configured to ensure high levels of cardiorespiratory load, anaerobic glycolytic energy contribution and high

threshold motor unit engagement is achieved. These factors are heavily reliant on the intensity threshold initiated via the work interval duration and or accumulating a certain amount of exercise time per session at training intensities at or above an athlete's VO_{2max}. Consequently, programming factors such as appropriate work interval intensity and duration, the number of intervals performed per session, and the specificity of exercise mode will dictate the short and long term physiological and performance responses expected of a given HIT intervention.

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CHAPTER 3: CHANGES IN STRENGTH AND POWER OF PROFESSIONAL RUGBY UNION PLAYERS OVER A PLAYING AND TRAINING SEASON

3.1 Introduction

Strength and power are key requirements for performance in a variety of athletic tasks. Rugby union players require maximum and explosive force production capabilities for both contact (e.g. scrummaging, rucking and mauling) and sprinting aspects of the game (Roberts et al., 2008, Tillin et al., 2013). Maximizing long-term adaptation of muscular force via resistance training is therefore one of the main goals of professional rugby players' conditioning programmes.

Most evidence on training adaptation arises from short-term studies (6-12 weeks) with sub elite athletes, where improvements in muscular strength and power are achieved with relative ease (Harris et al., 2000, Cormie et al., 2007a). These studies may not reflect the adaptation potential of well-trained rugby athletes where the concurrent training environment, the principle of diminished return, and the time-limited nature of a competitive season are all factors which make athletic development harder to achieve (Argus et al., 2009, Cormie et al., 2010b, Crewther et al., 2013). Consequently, a greater knowledge of the strength and power characteristics (e.g. enhancement, retention or decay) associated with the different phases of a professional season (e.g. pre-season, early-season, late-season) is required if effective and efficient resistance training strategies are to be developed.

The pre-season conditioning phase is considered a critical training period for professional rugby athletes. The aim of this phase is to develop key physical characteristics (e.g. strength, power, speed) whilst match play requirements remain low. Although research is limited, the pre-season training phase in professional rugby union has been demonstrated to initiate rapid improvements in measures of strength and body composition due to the greater volumes and frequencies of physical development training (Argus et al., 2010). During the competitive phase (in-season), physical conditioning volume may reduce whilst rugby specific training volume increases. During the in-season period, the goal of the conditioning programme is to maintain the physical gains made during the pre-season period. However, limited data currently exists tracking the extent and magnitude of strength and power adaptation resulting from resistance training conducted during pre and in-season phases.

A 13 week in-season monitoring study reported moderate increases in lower body strength (9%) and maintenance of upper body strength (-1%), with small decreases in lower and upper body power (both -3%) (Argus et al., 2009). Over a two-year period in elite rugby union players, strength capacity increased by 7 and 12% in upper and lower limbs respectively (Appleby et al., 2012). Similar findings have been reported in highly trained rugby league players, where two years of resistance training lead to a 6% increase in upper limb strength (Baker and Newton, 2006), whilst lower limb strength increased by 14% over a four-year period (Baker and Newton, 2006). Elite rugby athletes may not demonstrate extreme changes in physical performance capacity, but evidence does suggest that meaningful improvements in strength and power can be achieved when monitored over long-term training periods.

Identifying strength and power responses to longer-term training is therefore required if the full physical potential of elite rugby athletes is to be realised. Tracking chronic changes in strength (e.g. one repetition maximum [1RM] testing) and power exercise performance has been documented, but it is currently unknown how kinetic measures of strength alter over the course of a professional rugby union season. Analysis of peak force and early force development via maximum voluntary isometric squat testing have been shown acutely to reflect athletic performance potential in elite level rugby union players (Tillin et al., 2013). Isometric force testing has also been widely used to evaluate force generation capacity during both early (50-100 ms) and late (>100 ms) phase of muscle contractions that characterise maximal intensity athletic movements (Aagaard et al., 2002, Lima-Silva et al., 2013, Tillin et al., 2012). Consequently, force assessments appear to reflect the intrinsic muscle characteristics associated with athletic performance tasks (Aagaard et al., 2002), and may therefore be useful when elucidating the true potential of training practice during different phases of a professional rugby season.

When investigating the effects of a playing season on physical performance, it is important that the performance outcomes are viewed within the context of the professional environment. Whilst literature has attempted to track athletic variables across pre and in-season periods during southern hemisphere competitive structures (Argus et al., 2009, Appleby et al., 2012), no such evidence exists for elite rugby union athletes competing in the English premier division. The structure of the training and playing season is very different in southern and northern hemisphere rugby union, with 14 weeks of competition per season in the southern hemisphere (Argus et al., 2009; Appleby et al., 2012), compared with 34 weeks in the English premiership. This will influence the treatment effects (i.e. resistance training dosage) of resistance training protocols and subsequently alter athlete force development, retention and decay potential. To date, strength and power characteristics have not been reported over an extended period of time in elite players in England.

Hence, the purpose of this study was to investigate changes in strength and power over the different phases of a professional competitive season of rugby union in the English premier division. It was hypothesised that strength and power would improve over the pre-season period and that these increases would be maintained throughout the competitive season.

3.2 Materials and methods

3.2.1 Experimental Design

This longitudinal tracking study investigated markers of strength and power in professional rugby union athletes over 45-weeks. Athletes were assessed on four separate occasions: initial testing (weeks 1-3; [T1]) which represented the start of the pre-season period, at the end of the pre-season period (weeks 10-12; [T2]), mid-way through the competitive season (weeks 31-33; [T3]) and at the end of the competitive season (weeks 43-45; [T4]). All testing sessions were performed in the morning between 08:00 and 10:00 hours prior to the athletes' first training session of the day. During each testing session an explosive leg press (power) was performed first followed, after a 5-minute recovery period, by an isometric squat (strength). Neither the isometric squat nor explosive leg press formed part of the athletes' training programme.

3.2.2 Participants

Twenty two professional rugby union athletes from an English premiership professional rugby team volunteered to take part in this study. The participants competed in both the English premiership and European cup competitions from September 2013 to May 2014. Three of the participants also performed at senior international level. Each athlete had at least two years of resistance training experience. Each participant was over the age of 18 and provided written informed consent. Six participants were removed from analysis due to failure to complete physical assessments at all time points due to match related injury. Subsequently, data for sixteen participants (age, 23 ± 4 years; height, 1.89 ± 0.10 m; body mass, 109.0 ± 11.4 kg) were analysed. Study procedures were approved by an institutional ethics committee (Research Ethics Advisory Committee for Health, University of Bath).

3.2.3 Training

The volume load and number of lower body strength training, speed-power training, rugby training, matches, total running distances, high intensity and sprinting running distances and number of collisions each week was dependent on the training phase and is outlined in Table 3.1. The strength training programme was periodised throughout the year and was designed to develop maximal and explosive force production qualities. Strength training programmes were

prescribed to each athlete based on the individual's physical limitations, positional requirements and injury history. The types of strength training performed throughout the season are presented in Table 3.2. Throughout each season phase, the acute and chronic implementation of strength and power training programme prescription was implemented to control for the potential interference effects of the concurrent environment. During the first 6 weeks of the pre-season phase strength and power training was performed on days when no rugby training or endurance training occurred. During the second six weeks of pre-season and during in-season training periods, strength and power training was performed on days when rugby volume was low or on training free days (i.e. no rugby or endurance training). These strategies were implemented to ensure the adaptations associated with strength training (i.e. muscle protein synthetic response pathways) were not repressed due to the activation of molecular signalling pathways responsible for endurance adaptation (Hawley, 2009). The structuring of the strength and power training order in this manner throughout the season also ensured that each athlete had the widest recovery window possible during a weekly training cycle to optimise the adaptation potential of the training stimulus.

Table 3.1 Average weekly total volume load for lower limb resistance training, average number per week of lower limb resistance training, speed and power training, rugby training sessions and matches played as well as average weekly running volumes, high intensity and sprinting running volume (i.e. total volume >5 m/s) and the average number of total weekly collisions (mean ± SD).

	Training phase		
	T1-T2 (weeks 1-12)	**T2-T3** (weeks 13-33)	**T3-T4** (weeks 34-45)
Lower limb resistance training volume load (kg)	9110 ± 2335$^{*\#}$	5737 ± 2475	5662 ± 224
Lower limb resistance training	1.6 ± 0.1$^{\#}$	1.4 ± 0.6	1.3 ± 0.4
Speed-power training	1.1 ± 0.1$^{*\#}$	0.4 ± 0.1	0.4 ± 0.2
Rugby training	4.1 ± 0.8	4.2 ± 0.2	4.3 ± 0.4
Matches	0.2 ± 0.1$^{*\#}$	0.5 ± 0.1	0.7 ± 0.1$^{\neq}$
Running volume (m)	13201 ± 2924	11852 ± 3023	10384 ± 3570
High intensity running & sprinting volume (m)	513.9 ± 200	519.7 ± 248	526.7 ± 235
Collision volume	36.8 ± 29.3	40.8 ± 22.7†	26.7 ± 12.6

*T1-T2 significantly different to T2-T3 $p < 0.001$
$^{\#}$T1-T2 significantly different to T3-T4 $p < 0.001$
$^{\neq}$T3-T4 significantly different to T2-T3 $p < 0.05$
†T2-T3 significantly different to T3-T4 $p < 0.05$
Note: volume load (kg) = number of sets × number of repetitions × weight lifted (kg) (Peterson et al., 2011)

Table 3.2 Typical lower body strength training session design performed over the course of the season.

Training adaptation	Contraction mode	% 1RM	Sets	Reps
Maximal-strength	Isotonic	89-95	4-6	2-4
Hypertrophy-strength	Isotonic	75-89	4-6	4-10
	Eccentric	110-130	4-6	4-6
1 RM = 1 repetition maximum				

The pre-season phase (weeks 1-12), was split into two six week training blocks. The first five weeks were characterised by a greater frequency and load volume of hypertrophy-strength training (both isotonic and eccentric), power (i.e. 4-5 sets of 4-6 reps at 30-70% 1RM) and speed (i.e. 4-6 reps of flat sprints and or 4-6 reps of sled accelerations at 10-30% body mass load) with a lighter week being the last week of the cycle. The aim of the first pre-season cycle was to develop muscle size, strength, power and speed. The last six weeks of this phase were characterised by four weeks of high volume load maximal-strength training (isotonic focus), power and speed training, with the last two weeks programmed as recovery weeks (i.e. volume load of maximal-strength, power and speed training reduced). The aim of the second pre-season cycle was to develop maximal-strength, power and speed. During the first competition phase (weeks 13-33), hypertrophy-strength (isotonic) and maximal-strength sessions were cycled in five weekly blocks with a lighter week being the last week of each cycle. Power and speed training was performed during both blocks. Volume load during this period was reduced in comparison to pre-season for both hypertrophy-strength (i.e. 75-85% 1RM, 3-5 working sets, 4-8 reps per set), maximal-strength (89-92% 1RM, 3-4 working sets, 2-4 reps per set), power (i.e. 3-4 sets of 4-6 reps at 30-60% 1RM) and speed (3-4 reps of flat sprints and or 3-4 reps of sled accelerations at 10% body mass). The aim of this cycle was to develop or maintain strength, power and speed qualities. The second competition phase (week 34-35) was characterised as strength, power and speed maintenance. During this phase hypertrophy-strength and maximal-strength sessions were cycled in five weekly blocks with the last two weeks characterised as lighter weeks (i.e. reduced total weekly load volume) with power and speed performed during both cycles. All training programmes were individually designed and prescribed within the context of progressive overload. Whilst the aims of each training phase have been outlined, variability existed in the outcome potential of the participants due to the different match play demands which characterised the range of athletes selected for analysis. Consequently, some individuals may have experienced a greater in-season opportunity to

develop physical qualities whilst other athletes focused on maintaining during in-season phases.

Table 3.3 lists typical multi-joint lower body strength, power and speed exercises used during training sessions. Supplementary isolation exercises for the calf, hamstring and gluteal muscle groups were prescribed after the completion of multi-joint exercises. Each multi-joint lower body exercise began with three sets of progressively increasing load prior to three to six maximal intensity sets at the load prescribed for the specific strength training session.

Table 3.3 Typical strength, power, speed and plyometric exercises used during training.

Strength exercises	Power, speed and plyometric exercises
Belt squat	Horizontal leg drive (prowler)
Trap bar deadlift	Weighted jumps (unilateral, bilateral)
Leg press (supine, incline, unilateral, bilateral)	Horizontal jumps (unilateral, bilateral)
Hip Extensions (barbell, unilateral, bilateral)	Sled sprints (5-10 m)
Eccentric leg press (unilateral, bilateral)	Flat sprints (5-20 m)
Eccentric hamstring (unilateral, bilateral)	Change of direction drills
Calf raise	

Rugby training involved full team training with match simulation. These sessions incorporated varying levels of contact from tackle/static exertion simulation, to pads, to full contact. Position specific unit training was also performed weekly whereby skills, technique and tactics were practiced (i.e. scrum, lineout, backline play) based on each individual's positional demands. Energy system conditioning consisted of two high intensity interval training (HIT) sessions per week during the pre-season period (i.e. 15-30 minute total duration, repeated efforts of 5 to 20 second work periods, 1:2-4 work to rest ratio). Athletes were prescribed exercise intensities that corresponded to approximately 110-130% of their maximal aerobic speed. Conditioning sessions comprised predominantly running with changes of direction (e.g. 180°), however bike sessions were also used. Athletes also performed two sessions per week of small-sided games (30 minute duration). Due to the high running conditioning component of rugby training, conditioning sessions were reduced to one per week during the last 4 weeks of pre-season. During the first and second competition phases conditioning sessions were delivered on an individual basis dependent on weekly training and match play volume.

3.2.4 Test battery

Explosive leg press

Lower body power was monitored using an explosive leg press exercise performed on a bespoke leg press machine. The explosive leg press used a plate-loaded sled with padded shoulder supports positioned on a steel constructed frame with rails inclined at 24° (Figure 3.1). The sled was mounted on the rails on low friction steel rollers. The starting position of the sled was adjustable to the nearest 50 mm and the foot platform was gridded to ensure lower limb joint angles and starting foot positions were standardised. A linear position transducer analysis system (Gymaware, Kinetic Performance Technology, Canberra, Australia) was attached to the sled and utilised to record concentric mean power. The linear position transducer uses a tethered cord attached to the equipment (in this instance explosive leg press sled) to extract time-displacement data, and from this movement velocities and subsequent accelerations are calculated. This kinematic information is then differentiated to provide estimates of power when the mass of the load and participants on the equipment are factored in (Drinkwater et al., 2007, Crewther et al., 2011d). The Gymaware linear position transducer system has been demonstrated previously to provide moderate to strong (r = 0.59-0.87) relative and absolute validity in estimates of power when compared to criterion measures (i.e. force platform measures) (Crewther et al., 2011d). Equally, Drinkwater et al. (2007) demonstrated strong validity (CV = 1.0-3.0% and ICC = ≥0.97) in Gymaware measures of bilateral squat jump mean and peak power in comparison to cinematographic analysis (criterion measure). Subsequently, the Gymaware linear position transducer system is a valid method of collecting kinetic data on resistance training movements. Five repetitions were performed on the explosive leg press to ensure maximal power was achieved (Baker and Newton, 2007), with the best repetition used for analysis. During each repetition, participants were instructed to perform a triple extension movement with the intent to generate maximum velocity of the sled.

Testing for the explosive leg press was assessed with a load which represented 177% of the group mean body mass taking into consideration the effect of gravity on the incline plane. This was selected based on the mean percentage of body mass for which maximal mean mechanical power was attained during pilot work. More specifically, a group of elite rugby players (n = 16, age; 26 ± 2.5, height; 1.87 ± 0.1, body mass; 111.7 ± 10.3 kg), from the same team as the participants in the current study, performed explosive leg press movements against plate weight loads of 75, 100, 125, 150, 175 and 200% of body mass. The group mean load that elicited the highest mean mechanical power outputs was identified at 177 ± 13% of body mass. Consequently, during the present study the group mean body mass values at initial testing were multiplied by 1.77 and the subsequent value (192.9 kg) was added to the sled and inputted into the optical encoder system and utilised as the external resistance by which normalized mean

power was assessed at each testing session. Analysis of reliability was conducted with data from the five consecutive explosive leg press trials performed during initial testing (T1). The intraclass correlation coefficient (ICC) was 0.97, and the typical error (TE) for mean power was 2.3%. It was not possible to assess between-session reliability for power measures in the current study. However, using similar methods past studies have reported between-session reliability measures of mean power (ICC = 0.86; CV = 5.7%) (Harris et al., 2007).

A)

B)

Figure 3.1 Photograph of the custom built explosive leg press showing; A) participant positioning at the start of the movement, B) participant at extension during the jump.

Isometric squats

Isometric squats were completed on a squat rig that consisted of a horizontal barbell above a portable force platform (AMTI ACCUGAIT 0341, USA). The isometric squat was chosen over other measures of multi-joint force production (i.e. mid-thigh pull) as squats are widely used in athletic training for improving lower body force characteristics and the participants in the present study were all familiar with generating force through squat based movements. Equally the isometric squat has been used previously in well trained rugby athletes as a reliable measure of peak and early force monitoring in a practical applied setting (Tillin et al., 2013). Ground reaction forces were recorded by the force platform at a sample rate of 200 Hz. This sampling rate is appropriate for determining peak force as consistent and stable plateaus in the force-time curves were realised, however it is acknowledged that this sampling rate may reduce the accuracy by which early force measures can be assessed (Buckthorpe et al., 2012).

Zero force was defined as the participant's body weight. Participants stood on the force platform in a back squat position with a 20 kg horizontal barbell touching their posterior deltoids and middle trapezius (Figure 3.2). Peak force has been reported to occur at later knee flexion angles during the concentric phase of a squat (130-170°), therefore a knee angle of approximately 140° was used to measure peak force output (Cormie et al., 2007a). Consequently, bar height was set and produced an average knee flexion angle of 137.6 ± 3.7° as measured by a goniometer. Once bar height was established, metal stops were placed on top of the barbell to stop its upward motion. During all squat trials, participants were instructed to adopt a neutral spine position and maintain a stable posture throughout each contraction period. Participants performed two warm up trials, one at 60% and one at 80% of their perceived maximal exertion. They then performed three maximal isometric efforts where they followed a simple 5 second countdown, followed by "go, 1, 2, 3, 4, stop". During the contraction, each participant was instructed to push against the bar as fast and as hard as possible for the entire count, with 3 minutes of rest separating each trial (Blazevich et al., 2002). Knee angles and body alignment was checked before each trial and verbal encouragement was given to increase participant motivation.

Force data collected via the AMTI portable force platform were exported to Microsoft Office Excel. The vertical ground reaction force data was smoothed via a 10 point moving average. The greatest peak vertical ground reaction force of the three maximal squats was defined as peak force. As force production during the early phase of isometric squatting (<100 ms) has been reported to be associated with athletic performance in elite rugby players (Tillin et al., 2013), force at 50 ms and 100 ms intervals from the onset of force application was identified. To ensure the identification of force onset had repeatability, an onset threshold was used for all isometric squat contractions (Owen et al., 2014). The initiation threshold was defined as the instant, after the signal to initiate force had been given, that the vertical ground reaction force exceeded the mean plus or minus 3 SD of the quiet resting value. Each signal onset was also manually checked by visual identification by the same investigator for all early force onset measurements. Peak force, force at 50 ms and force at 100 ms was reported in absolute terms and then normalised to body mass by allometrically scaling with an appropriate power value (i.e.N.kg $^{-0.66}$ [Folland et al., 2008]). To account for the body size differences of professional rugby players, the normalised data were utilised for further analysis (Crewther et al., 2009a). Analysis of reliability was conducted with data from the three consecutive isometric squat trials performed during initial testing (T1). For measures of peak force, the ICC was 0.95, and the typical error was 2.4%. The measure of force at 50 ms reported an ICC of 0.53 and a TE of 9.6%, whilst force at 100 ms reported an ICC of 0.51 and a TE of 11.2%. It was not possible to assess between-session reliability for early force measures in the current study. However, past studies have reported between-session reliability measures of force at 50 ms (ICC = 0.80; CV

= 16.6%) and force at 100 ms (ICC = 0.91; CV = 6.4%) (Buckthorpe et al., 2012).

Figure 3.2 Participant position for the isometric squat.

3.2.5 Statistical analysis

All training session data, force and power values are represented as Mean ± SD. A one-way ANOVA with repeated measures was used to identify if significant differences existed over the three training phases between the training session data collected (Table 3.1). All training session data was tested for sphericity and paired t-tests were used for post hoc analysis. Because of the elite nature of the participants, changes in measures of force and power were analysed according to the concept of smallest worthwhile change through effect size (ES) statistics (Hopkins, 2004). This procedure has been used in previous studies tracking long-term performance changes in elite rugby athletes (Argus et al., 2009, Baker, 2013). The smallest worthwhile change is a reference value (calculated as 0.2 of the pooled between-participant standard deviation) that permits the calculation of the probability that an observed change in performance is large enough to have an important effect on performance in team sport athletes (Hopkins, 2004). This statistical methodology has been advocated as a more appropriate strategy for identifying meaningfulness of change in elite athletes since well-trained individuals display smaller alterations in performance than less trained populations (Hopkins, 2004, Winter et al., 2014). Consequently, small changes in elite athlete performance may not

reach statistical significance despite being practically worthwhile in the competition environment (Winter et al., 2014).

Effect size statistics were calculated for the magnitude of difference in the force and power scores between three different stages of the season (initial testing vs. post pre-season [T1-T2], post pre-season vs. mid-season [T2-T3], mid-season vs. end of season [T3-T4]) and between the start and end of season measures (initial testing vs. end of season [T1-T4]). Changes in force and power were categorised as trivial (ES <0.2), small (ES 0.2-0.6), moderate (ES 0.6-1.2), large (ES 1.2-2.0) and very large (ES >2.0) (Hopkins, 2004). In order to identify the uncertainty around the true value of the reported effect size statistics, 90% confidence intervals around each effect size were reported based on the methods of Hopkins (2007). Subsequently, magnitude based clinical inferences were attached to each effect size and reported as the percentage chance, with descriptor, that the true effect was beneficial, trivial or harmful (Hopkins, 2007). For an effect size to be clear, the odds of benefit relative to odds of harm (odds ratio) had to be > 66 (Hopkins, 2007).

3.3 Results

Between initial testing to post pre-season (T1-T2), the change in peak force was unclear (+2.7%, ES = 0.21 ± 0.30, Table 3.4), whilst very likely beneficial increases in force at 50 ms (+16.0%, ES = 0.75 ± 0.40, Table 3.4) and 100 ms (+13.9%, ES = 0.63 ± 0.40, Table 3.4) were observed. The change in power was likely trivial (+0.2%, ES = 0.02 ± 0.20, Table 3.4) (Figure 3.3A).

Between post pre-season to mid-season (T2-T3) there was an unclear change in peak force (+1.9%, ES = 0.20 ± 0.40, Table 3.4), and a likely beneficial increase in power (+3.6%, ES = 0.31 ± 0.20, Table 3.4). Changes in force at 50 ms (+1.6%, ES = 0.09 ± 0.40, Table 3.4) and 100 ms (+2.8%, ES = 0.16 ± 0.40, Table 3.4) were unclear (Figure 3.3B).

Mid-season to end of season (T3-T4) there was a likely harmful decrease in force at 50 ms (-6.3%, ES = -0.39 ± 0.30, Table 3.4) and 100 ms (-8.8%, ES = -0.52 ± 0.40, Table 3.4). Changes in peak force (-0.6%, ES = -0.07 ± 0.50, Table 3.4) and power (-0.8%, ES = -0.08 ± 0.50, Table 6.4) were trivial (Figure 3.3C).

When performance was analysed between initial testing and end of season testing (T1-T4), increases in peak force (+4.1%, ES = 0.32 ± 0.40, Table 3.5), power (+3.0%, ES = 0.29 ± 0.30, Table 3.5) and force at 100 ms (+6.9%, ES = 0.32 ± 0.40, Table 3.5) were possibly beneficial,

whilst force at 50 ms (+10.5%, ES = 0.55 ± 0.40, Table 3.5) was likely beneficial (Figure 3.3D).

Table 3.4 Percentage change and effect size (ES) with 90% confidence intervals (CI) in early and peak force and power between each test during the 45 week season. T1= baseline; T2 = post pre-season, T3 = mid-season, T4 = end of season, ES = effect size.

	T1-T2				T2-T3				T3-T4			
	% Change	ES	ES (90% CI) Lower	ES (90% CI) Upper	% Change	ES	ES (90% CI) Lower	ES (90% CI) Upper	% Change	ES	ES (90% CI) Lower	ES (90% CI) Upper
PF ($N.kg^{-0.66}$)	2.7	0.21	-0.1	0.5	1.9	0.20	-0.2	0.6	-0.6	-0.07	-0.4	0.5
Power (W)	0.2	0.02	-0.2	0.2	3.6	0.31	0.1	0.5	-0.8	-0.08	-0.1	0.3
F50 ($N.kg^{-0.66}$)	16.0	0.75	-0.3	1.2	1.6	0.09	-0.3	0.5	-6.3	-0.39	-0.7	-0.1
F100 ($N.kg^{-0.66}$)	13.9	0.63	0.3	1.0	2.8	0.16	-0.2	0.5	-8.8	-0.52	-0.9	-0.1

PF = Peak force, F50 = force at 50 ms, F100 = force at 100 ms

Table 3.5 Percentage change and effect size (ES) with 90% confidence intervals (CI) in early and peak force and power between the end of season test (T4) and the baseline test (T1).

	T1-T4			
	% Change	ES	ES (90% CI)	
			Lower	Upper
PF (N.kg$^{-0.66}$)	4.1	0.32	-0.1	0.7
Power (W)	3.0	0.29	0.0	0.6
F50 (N.kg$^{-0.66}$)	10.5	0.55	0.1	1.0
F100 (N.kg$^{-0.66}$)	6.9	0.32	-0.1	0.7

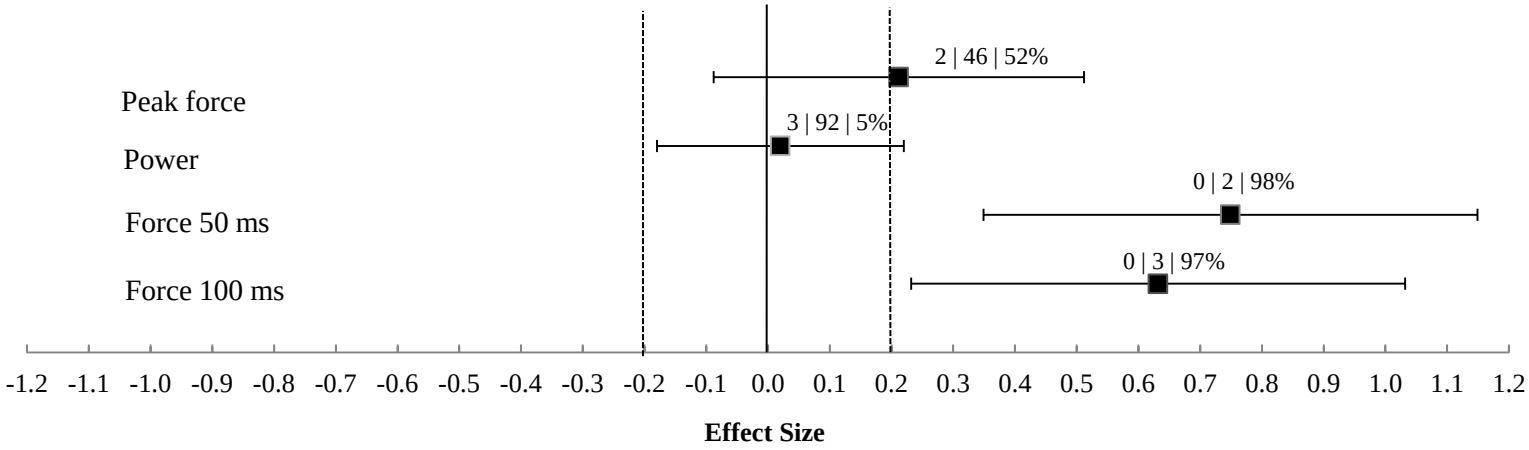
PF = Peak force, F50 = force at 50 ms, F100 = force at 100 ms

Table 3.6 Values of early and peak force, power and body mass (mean ± SD) at each of the four testing periods over the competitive season. T1= baseline; T2 = post pre-season, T3 = mid-season, T4 = end of season.

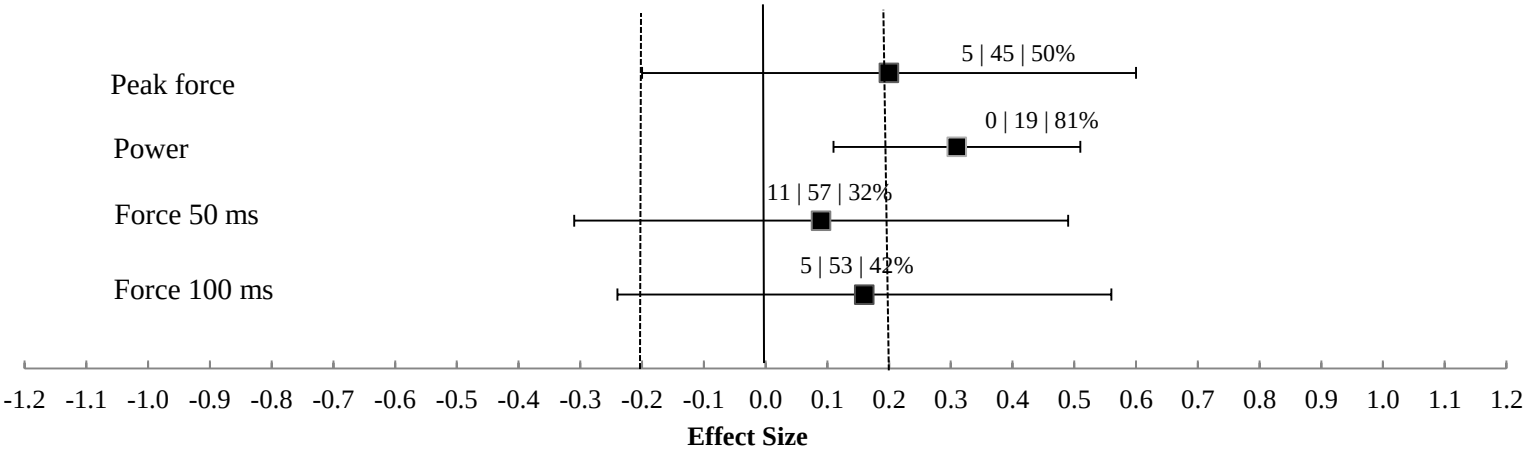
	T1	T2	T3	T4
PF (N.kg$^{-0.66}$)	158.5 ± (23)	162.8 ± (17)	165.9 ± (13)	164.9 ± (17)
Power (W)	1197.2 ± (134.0)	1200.1 ± (153.2)	1243.2 ± (126.2)	1233.4 ± (117.6)
F50 (N.kg$^{-0.66}$)	81.2 ± (14.9)	94.2 ± (19.5)	95.7 ± (14.6)	89.7 ± (16.2)
F100 (N.kg$^{-0.66}$)	95.6 ± (22.6)	109.0 ± (19.7)	112.1 ± (19.3)	102.2 ± (18.7)
Body mass (kg)	109.0 ± (11.4)	109.9 ± (10.7)	110.3 ± (10.9)	110.3 ± (11.1)

PF = Peak force, F50 = force at 50 ms, F100 = force at 100 ms

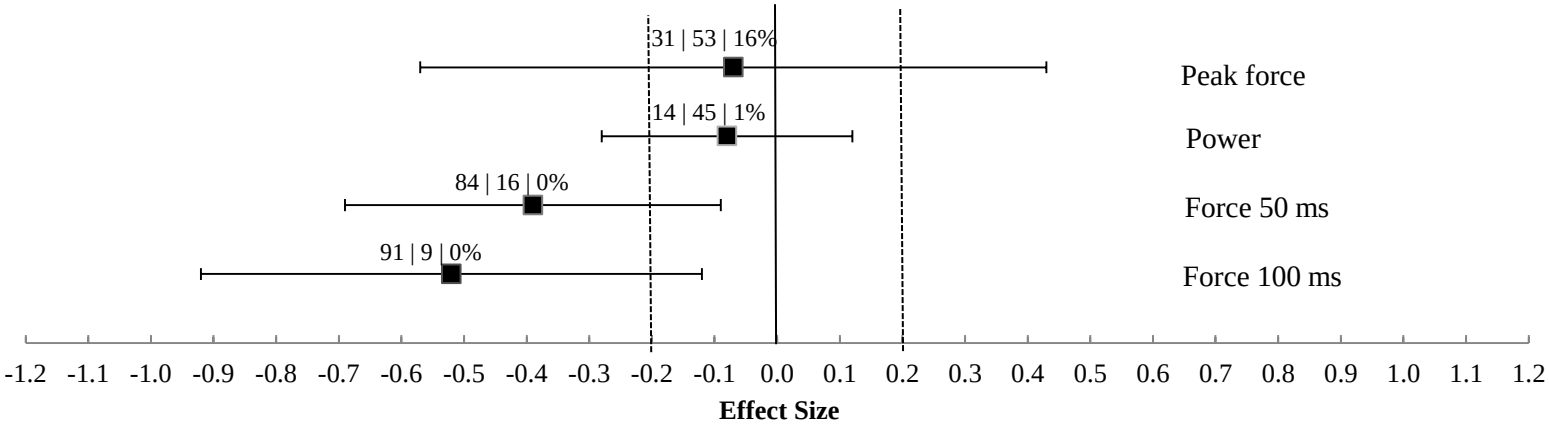
A)


2 | 46 | 52%
Peak force
3 | 92 | 5%
Power
0 | 2 | 98%
Force 50 ms
0 | 3 | 97%
Force 100 ms
-1.2 -1.1 -1.0 -0.9 -0.8 -0.7 -0.6 -0.5 -0.4 -0.3 -0.2 -0.1 0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0 1.1 1.2
Effect Size

B)


5 | 45 | 50%
Peak force
0 | 19 | 81%
Power
11 | 57 | 32%
Force 50 ms
5 | 53 | 42%
Force 100 ms
-1.2 -1.1 -1.0 -0.9 -0.8 -0.7 -0.6 -0.5 -0.4 -0.3 -0.2 -0.1 0.0 0.1 0.2 0.3 0.4 0.5 0.6 0.7 0.8 0.9 1.0 1.1 1.2
Effect Size

C)



D)

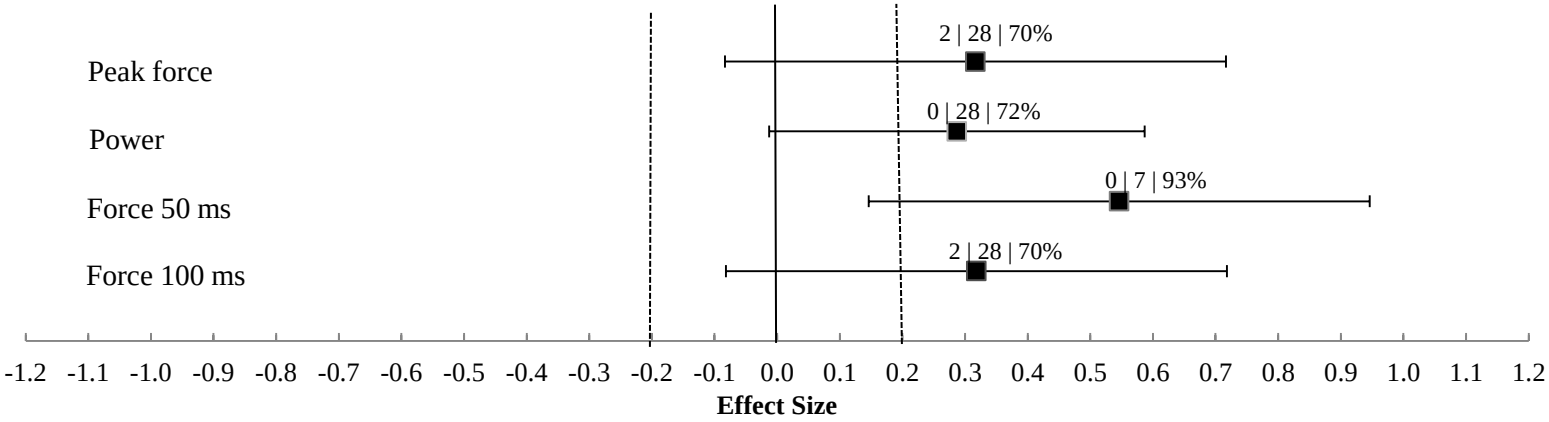


Figure 3.3 Effect sizes (with 90% CI) for the differences in each performance measures at: A) initial testing vs. end of pre-season (T1-T2), B) end of pre-season vs. mid-season (T2-T3), C) mid-season vs. end of season (T3-T4), and D) initial testing vs. end of season (T1-T4). Data labels give likelihoods that effect is substantially harmful | trivial | beneficial.

3.4 Discussion

This investigation tracked strength and power characteristics in professional rugby union athletes during a full competitive playing season. Beneficial improvements in early force production were reported after the pre-season training period, whilst beneficial increases in power were observed at mid-season testing. Peak force and power characteristics were maintained during the latter stages of the season. Over the 45 week training and playing period (T1-T4), beneficial increases in peak and early force production as well as power were identified, suggesting improvements in muscular performance can be achieved over a season of concurrent training and match play.

The 12-week pre-season period (T1-T2) resulted in the greatest magnitude of within season change in strength. Force at 50 ms and 100 ms demonstrated very likely positive improvements whilst possible beneficial increases in peak force were reported. This finding supports previous evidence demonstrating the positive effects of a pre-season training block on measures of lower body strength in professional rugby union athletes (Argus et al., 2010) and reflects the greater opportunity pre-season presents in team sports for physical preparation and in particular strength development (Argus et al., 2009, Comfort et al., 2012). In the present study, significantly greater lower limb resistance training volume loads were recorded during the pre-season when compared to mid and end of season phases whilst a significantly greater frequency of resistance training was completed during this period in comparison to the end of season phase (Table 3.1). Pre-season training also facilitates the implementation of more effective periodisation strategies. For example, strength training in the current study was a prioritised training goal and the timing and sequencing of sessions were dictated to avoid the interference effects of concurrent training (i.e. strength and endurance training were performed on separate days) (Sale et al., 1990, Baker, 2001a). The 12-week pre-season period also involved the lowest volume of matches played per week which limited the likelihood of injury (Williams et al., 2013) and reduced the holistic effects of fatigue which has been reported to decrease the potential for strength adaptation (Argus et al., 2009, Gabbett, 2005).

Power did not change during pre-season training (T1-T2). Similarly, Argus et al. (2010) reported small declines in lower-body power over a four week pre-season training cycle to be associated with the greater total training volumes accumulated during this period. Consequently, the elevated resistance training volume load during pre-season (Table 3.1) may have compromised the development of power. Power did however increase between the end of pre-season and mid-season testing (T2-T3), which was characterised by a reduction in total resistance training volume load. Mid-season testing was characterised by the highest peak force value when considered across the four testing periods (Table 3.6). Long-term changes in

power have been reported to be heavily reliant on changes in strength and multi-joint isometric peak force is a strong predictor of multi-joint dynamic performance (Baker, 2013, Beckham et al., 2013). At mid-season testing a significant moderate association was demonstrated between peak force and power ($r = 0.50$, $p = 0.02$). The elevated isometric peak force capacity of the athletes should therefore have supported the observed improvements in power witnessed at mid-season testing.

From mid to end of season testing (T3-T4) peak force and power remained unchanged. These findings support previous long-term analysis suggesting physical qualities such as strength and power can be maintained throughout in-season periods (Argus et al., 2009, Baker, 2001a, Gabbett, 2005). This training phase also reported small decreases in force at 50 and 100 ms. The maintenance of force and power and the reported decline in early force production during the latter stages of the season may reflect the diminishing scope for positive physical adaptation that can be expected in elite team sport athletes during this period (Baker, 2013).

Weekly rugby training volume, running distance, high intensity and sprinting running volume was not statistically different throughout each season phase, whilst match play volume was significantly higher during the final phase of the season in comparison to pre and mid-season phases (Table 3.1). Consequently, the potential for recovery between match play and weekly training may be particularly compromised during the final weeks of a season. Residual fatigue built up over the course of the season combined with inadequate recovery time between weekly competition and rugby training may have limited the effectiveness of strength and power training during this period and thus reduced the likelihood for positive physical adaptation to occur (Gabbett, 2005).

Clear beneficial but small effect sizes (ES range = 0.29-0.55) were reported for all the performance measures between T1-T4. The most robust training responses were therefore identified when changes in strength and power were analysed over the entire 45-week season. This supports current evidence that strength and power appear to be trainable in elite-football code athletes over competition periods (McMaster et al., 2013). In particular, the 4% (ES = 0.32) increase in peak force demonstrated comparable effect size changes to the 11% (ES = 0.50) (Appleby et al., 2012) increase in back squat 1 RM performance and is greater than the 1% (ES = 0.07) (Barr et al., 2014) increase in front squat 1 RM performance observed over one year in southern hemisphere rugby union athletes. Lower limb extension power also demonstrated a beneficial 3% (ES = 0.29) increase at the end of season. Previous analysis into power development appears to be more variable. Argus et al. (2009) identified a decrease (-3%, ES = <0.20) in jump squat power, whereas Barr et al. (2014a) reported a 7% (ES = 0.60) increase in relative power clean performance over a one year period in a similar cohort of rugby union athletes. The discrepancies in long-term strength and power development within

elite rugby research may reflect differences in the testing measures used (i.e. isometric force vs. maximum dynamic strength), the weekly prescription of training loads, volumes, frequencies and exercise patterns (i.e. jumps vs. Olympic lifting), and the competitive structures between professional environments (i.e. pre and in-season duration).

This is the first study to identify long-term changes in markers of peak and early force capacity in an elite applied environment since muscular performance tracking has typically been used in short-term non-elite training studies (Aagaard et al., 2002, Cormie et al., 2007a). In particular, (Tillin and Folland, 2014) demonstrated significant increases in maximum voluntary isometric force production (21 ± 12%), force at 50 ms (70 ± 77%) and force at 100 ms (16 ± 14%) in response to four weeks of strength training in recreationally active participants. The present study observed smaller increases in these values over longer training periods which may reflect the diminished central nervous system and hypertrophic adaptation potential reflective of elite athlete populations (Hakkinen, 1989, Cormie et al., 2010b). Nevertheless, beneficial increases in isometric peak and early force production combined with enhanced isotonic power suggests the current training effects support transference to dynamic rugby tasks. For example, the ability to explosively utilise high peak ground reaction forces during short (50-150 ms) time intervals is important for the development of stride length during acceleration (Barr et al., 2014a, Tillin et al., 2012, Weyand et al., 2000). High rates of force development across a multitude of contraction time scales are also a critical component for contact situations such as scrum engagements, collision effectiveness and rucking and mauling tasks (Quarrie and Wilson, 2000, Cazzola et al., 2014, Crewther et al., 2009a).

3.4.1 Limitations

The present study acknowledges the inherent limitations associated with tracking physical performance in applied sport settings. Whilst robust inferences can be made as to the changes in selected measure of physical performance, it must also be recognised that a level of uncertainty in some of the effect size changes did exist. Some of the athletes may therefore have demonstrated no clear improvements or small decrements in force or power at a given testing point. The variability in physical response patterns reported throughout each season phase may be reflective of the differing match play and rugby training load volumes experienced by the current athletic cohort. Not all participants were selected to perform in the same number of matches as other participants. Consequently, those who played in more games may have experienced lower opportunity for physical development due to reduced recovery time periods between match play, rugby training and strength training sessions. Individualised response patterns are also to be expected in applied team sports since factors such as injury

occurrence and adaptive potential to a training stimulus will be variable between individual athletes.

Interpretation of the change in force at 50 ms and 100 ms must also be approached with caution due to the high typical errors around the measurements. This may have been associated with the sampling rate of the portable force platform (200 Hz). This piece of equipment was partly selected due to the practicalities of administration within the applied rugby training environment. However, the relatively low sampling rate may have reduced the reliability in the measures of early force due to decreased resolution when identifying force onset. The effects of measurement and biological noise associated with the testing protocol may therefore limit the conclusions that can be made regarding the training induced changes in early force production. Future applied tracking research should look to include higher sampling rates whilst also identifying methods for reducing the noise associated with measurement of early force production. Finally, the use of a fixed (absolute) load during testing for the explosive leg press meant each participant generated force at different relative external resistances (i.e. not all participants worked against a load relative to 177% of body mass). The range of intensity by which the participant worked against was reported as 51 kg. This may have impaired the potential for maximum power development as some participants may have performed the explosive leg press with high external mass which may have resulted in a greater proportional decrease in velocity. Conversely, some participants may have performed the explosive leg press with lighter loads that entailed greater velocities but may have reduced power due to greater proportional decreases in external mass.

3.5 Conclusions

The net effect of an entire training and playing season on markers of strength and power in senior English premiership rugby players appears positive. The greatest opportunity for physical development occurs during pre to mid-season phases whilst maintenance to slight decreases in these qualities appears to characterise the latter stages of a season. Meaningful increases in these measures over a competitive season could support functional improvements in dynamic rugby tasks such as initial acceleration, collision effectiveness and static exertion activities (e.g. scrum, ruck and maul).

3.6 Practical applications

Approximately 1-2 lower body strength and power training sessions performed weekly with high relative exercise intensity may be an appropriate strategy for developing and maintaining the force and power characteristics which underpin physical performance for professional rugby union. If further gains in strength and power are to be achieved during the final stages of

a season, then a reduction in external rugby training load is needed to limit the effects of residual fatigue and to maximise resistance and power training response pathways. Finally, if strength and power training is appropriately sequenced and periodised during pre and in-season phases, then meaningful adaptation in these qualities can occur in conjunction with the concurrent training loads which characterise a competitive season of English premiership rugby.

CHAPTER 4: EFFECTS OF A COMBINED STRENGTH AND POWER (COMPLEX) TRAINING INTERVENTION ON ATHLETIC PERFORMANCE AND HORMONE CONCENTRATIONS IN ELITE RUGBY UNION PLAYERS DURING AN IN-SEASON PERIOD

4.1 Introduction

The development of strength, power and speed is considered a primary contributor to successful performance in rugby union, where the demands of the sport rely heavily on the execution of high force and velocity based movements (Roberts et al., 2008). Professional rugby union also requires a multifaceted training approach which, in a time-limited environment, can concurrently combine enhancement of physical capacity across several domains (e.g. strength, power, endurance) alongside sport-specific training in order to improve competition outcomes.

Acute enhancement of speed and power has been identified in athletes following the execution of a biomechanically similar high intensity (> 80% 1 repetition maximum [RM]) weightlifting exercise (Chiu et al., 2003, Hodgson et al., 2005, Bevan et al., 2010b, Kilduff et al., 2008). The performance enhancement associated with contrasting resistances is attributed to a condition known as 'post-activation potentiation' (PAP). Proposed mechanisms for the PAP effect include increased neural activation of higher threshold motor units (Trimble and Harp, 1998), and enhanced actin-myosin cross-bridging cycling rates (Hodgson et al., 2005). Combining strength and power complexes into a training programme has been referred to as 'complex training', which has emerged as a popular modality in the applied team sport environment as a time-efficient method for developing both strength and power (Docherty D, 2004).

The effectiveness of complex training programmes for athletic performance in elite team sport athletes has not been clearly demonstrated. Comyns et al. (2010) identified a significant training effect for 30 m sprint performance when sprints were preceded by heavy [3 RM] back squatting in five complex training sessions, but the study lacked a control training group. The effects of complex training on strength characteristics have also yet to be established. Specifically, no study to date has investigated whether the high intensity weightlifting component in a complex set designed to improve power also provides an adequate stimulus for strength to be maintained or developed.

Acute and chronic changes in hormonal concentrations have previously been purported to be markers of adaptive physiology to resistance training due to the potential, but contentious, role of testosterone and cortisol in influencing protein turnover and skeletal muscle growth (McCaulley et al., 2009b, Smilios et al., 2003). Acute stimulation of endogenous hormones may also impact neuromuscular functioning, potentially through short-term neural and intracellular pathways (Blanco et al., 1997, Estrada et al., 2003, Hamdi and Mutungi, 2010b). These non-genomic hormonal effects may have implications for the execution of strength, power and speed training within elite team sport environments (Cardinale and Stone, 2006, Crewther et al., 2009c, Crewther et al., 2011a). To date, there is limited research assessing acute exercise responses and chronic changes in baseline concentrations of testosterone and cortisol in response to training programmes that incorporate both strength and power exercises in concurrent athletic environments, in order to identify if hormones have a role in adaptation in elite athletes.

The primary aim of this study was to investigate the effects of a complex training programme on baseline measures of muscular strength, physical performance (i.e. power and speed) and body composition in elite professional rugby players who were training and playing concurrently during a mid-season phase. This training phase was selected as it represents an important time in a professional season when strength and power development must be maximised if physical capacities are to be maintained during the final stages of the season (as demonstrated previously in Chapter 3). The secondary aim was to analyse the effects of complex training on acute alterations (pre to post exercise) and baseline changes (pre to post training) in testosterone and cortisol concentrations. Based on evidence from acute studies, it was hypothesised that repeated execution of a complex training protocol would provide an effective stimulus for improvements in baseline measures of strength and performance. It was also hypothesised that any alterations in baseline strength and performance would be associated with training-induced changes in baseline hormones. The intended outcome from the study was to provide coaches and sport scientists with a better understanding of the efficacy of the complex training modality during an applied training period in elite team sport athletes.

4.2 Materials and methods

4.2.1 Experimental design

A crossover design was used to assess the effects of a complex training intervention on measures of strength, power, speed, body composition and hormones in elite rugby players. Participants performed a complex training (‘complex’) and a strength training (‘strength’) intervention in two 4-week training blocks separated by a 2-week recovery period (Figure 4.1).

The strength intervention was the control condition. During the 2-week recovery period, participants abstained from all structured training and competition loading. Performance was assessed at four time points: pre- and post-training block one, and pre- and post-training block two. The allocation of participants to the order of training condition was randomised using the 'rand' function in Microsoft Excel. Training sessions for each intervention were performed twice a week at the same time of the day (4 PM ± 1 hour) to control for circadian variation (Kraemer et al., 2001).

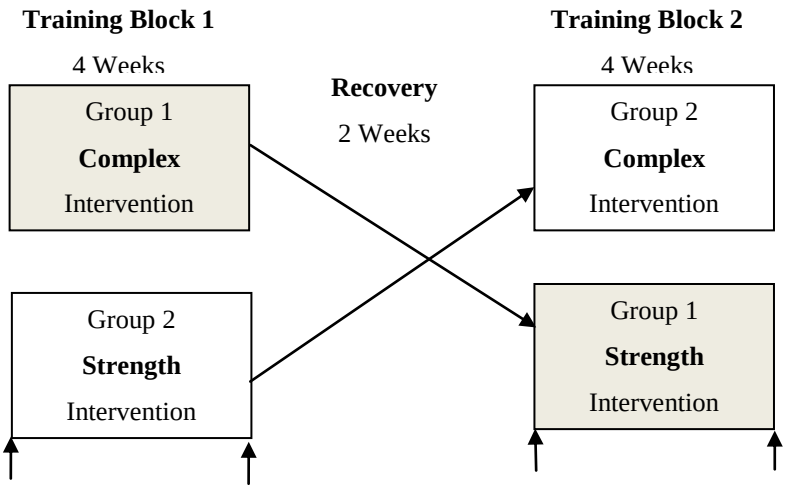


Figure 4.1 Schematic for the cross-over experimental design of the study.

4.2.2 Participants

Fourteen young professional academy rugby union players each with a minimum of two years strength and power training experience participated in the study. Training interventions were carried out during the mid-phase of the competitive season. During the 10-week experimental period participants performed four sessions of rugby union training per week (except during the 2-week recovery period), that included metabolic conditioning, technical and skills based training and played in a total of four official games. All participants performed the same moderate volume and intensity weight-training programme prior to the initiation of the study to ensure similar training status at baseline. Each participant was over the age of 18 and provided written informed consent. Study procedures were approved by an institutional ethics committee (School for Health Research Ethics Panel, University of Bath). Participants had to complete ≥ 90% of the training sessions to be included in the study. Three participants were excluded from the analysis due to injuries incurred through competition and training, none of which were incurred as part of the study interventions. The final number of participants in the

study was 11 (mean ± SD: age 19.2 ± 0.7 years, mass 98.6 ± 10.6 kg, height 181.9 ± 5.8 cm, body fat 12.3 ± 3.3%).

4.2.3 Workout design

Both the complex and strength interventions incorporated a multi-set training design characterised by weekly progression in either exercise load or volume (Table 4.1). In the week prior to the commencement of the first training period, all participants were assessed for strength, from which all external loads were calculated and prescribed on a weekly basis based on the methods of Beachle (2000). External load was increased when a participant was able to perform the targeted number of repetitions at the current workload with good form. Before each training session participants performed a 10-minute warm up consisting of stretching and movement preparation exercises specific for the muscle groups activated during the training session. All participants performed the intervention training sessions on the same day, at least two days after a game to minimize the effects of rugby competition. The intervention sessions were performed as the last exercise session of the training day (i.e. after rugby training), whilst each session was followed the next day by either a recovery day or a light training day (i.e. a low volume team practice session). The ordering of the interventions in this manner was an attempt to control the interference effects associated with the in-season concurrent training environment. As no other training was performed after the interventions, it was assumed the adaptive response pathways initiated via the strength training stimulus would not have been limited by any subsequent exercise modalities (e.g. endurance training) that could have repressed the strength adaptation signal (Hawley, 2009). Equally, by performing the interventions in the afternoon prior to a recovery or light rugby day, it was assumed that each athlete was provided with a sufficient time frame (within the confines of a concurrent weekly training structure) for an adaptive muscular response pathway to be optimised over the following 24 hour period when sustained rates of muscle remodelling may occur (Churchward-Venne et al., 2012). The volume and intensity of both training protocols has previously been reported to provide a sufficient stimulus to initiate strength development in highly trained athletes (Peterson et al., 2005). Each strength exercise performed throughout both protocols was selected based on its potential to recruit large muscle mass. The resistance training exercises performed each week for both the complex and strength interventions are presented in Table 4.2.

Table 4.1 Description of prescribed resistance training for the *complex* training and *strength* training interventions during the 4 week training block.

	Complex					Strength				
Week	Exercise order		Sets	Reps	Intensity (% 1RM)	Exercise order		Sets	Reps	Intensity (%1RM)
1	A	Strength 1	3 - 4	6	83	A	Strength 1	4	6	83
	B	Power or Plyometric		5 or 6	40 or BM	B	Strength 2		8	79
2	A	Strength 1	3 - 4	5	86	A	Strength 1	4	5	86
	B	Power or Plyometric		5 or 6	40 or BM	B	Strength 2		6	81
3	A	Strength 1	3 - 4	4	89	A	Strength 1	4	4	89
	B	Power or Plyometric		5 or 6	60 or BM	B	Strength 2		8	79
4	A	Strength 1	3 - 4	3	92	A	Strength 1	4	3	92
	B	Power or Plyometric		5 or 6	60 or BM	B	Strength 2		6	81

RM = repetition maximum, BM = body mass

Table 4.2 Resistance exercises completed each week in session 1 and 2 for the complex training and *strength* training interventions.

	Complex			Strength	
Session	Strength 1	Power	Plyometric	Strength 1	Strength 2
1	Back squat	Jump squat		Back squat	Romanian deadlift
	Bench press	Bench throw		Bench press	Chin-ups (weighted)
2	Deadlift		Counter movement jump	Deadlift	Rear foot elevated split squat
	Bench pull		MB throw	Bench pull	Incline bench press

MB = medicine ball

4.2.4 Complex training and strength training interventions

The complex protocol was designed to develop maximal strength and power. Participants were instructed to execute each strength exercise with a controlled eccentric (3s) and maximal concentric tempo whilst all power and plyometric exercises were performed as quickly as

possible. The complex intervention incorporated the principles of PAP; after the execution of the primary strength exercise there was a four minute rest period before participants performed a biomechanically similar power or plyometric exercise (Table 4.2). This contrast method of one power or plyometric exercise after every strength exercise was repeated for each set. Although optimal rest period appears to be highly individual (Bevan et al., 2010b), four minutes has been reported to provide sufficient time for an appropriate balance between fatigue and potentiation in team sport athletes (McBride et al., 2005). Equally, four minutes of recovery was selected as this duration has been shown to be the earliest time period when muscle potentiation is enhanced after a pre-conditioning back squat exercise in elite rugby athletes (Bevan et al. (2010b). Whilst optimal recovery time periods of eight minutes have been demonstrated in elite rugby athletes (Kilduff et al., 2007), the four minute recovery period also provided a more appropriate rest duration to ensure repeated sets of the complex exercises could be completed within the session time period. The strength exercise intensities over the 4-week training period have also been reported to be of a sufficiently heavy load (>83% 1RM) to initiate a potentiated state (Docherty D, 2004).

The strength protocol was designed to develop maximal strength. Participants were instructed to execute each exercise with a controlled eccentric (3s) and maximal concentric tempo. In each session participants performed the primary strength exercise followed two minutes later by the secondary strength exercise with a two minute rest period between sets. This method of exercise prescription has been reported to provide sufficient overload and recovery for strength development in elite athletes (Beachle, 2000). The session duration for both training interventions was approximately 50 minutes.

4.2.5 Strength and power testing

Back squat and bench press exercises were selected to assess strength. These were performed using an Olympic barbell, a squat rack and a bench. On the four testing occasions, participants performed the back squat and bench press with increasing loads until a two to four RM was achieved in the last set. The 1RM lift was then estimated from a prediction equation (Beachle, 2000). This method of maximum strength assessment was selected for safety reasons and has been reported to be highly reliable (Crewther et al., 2009c). Power was assessed via a jump squat and bench throw which were performed on a smith machine using controlled eccentric and ballistic concentric movements. Concentric maximum average power was calculated using an optical encoder analysis system (Gymaware, Kinetic Performance Technology, Canberra, Australia) which has been reported to provide valid measures of kinetics during jump squat and bench throw movements (Drinkwater et al., 2007). Five repetitions were performed for both power exercises to ensure maximal power was achieved (Baker and Newton, 2007), with

the best repetition used for analysis. Testing for both the jump squat and bench throw was assessed with a resistance of 44 kg (jump squat) and 65 kg (bench throw), which represented 55% of the group mean 1RM system mass back squat and absolute mass bench press. Absolute loads for power testing has previously been shown to be more sensitive to intervention-induced changes in power than relative individual specific loads (Baker and Newton, 2007), and the resistance of 55% 1RM was selected as it has previously been reported to be a sufficient load to achieve maximum mean mechanical power outputs in the jump squat and bench throw exercises for trained athletes (Baker, 2001b). However, differences in the literature exist regarding the optimal load required for both mean mechanical power and peak power measurements. Whilst 55% 1RM was selected for the present study, it is also recognised that optimal power (both mean and peak power) loading in elite athlete's populations is variable. Relative loads lighter than 55% 1RM, for example 30% 1RM for bench throw exercises and 0% body mass for jump squat exercises, have also been demonstrated to provide sufficient resistance for elite rugby athletes to generate optimal peak power (Bevan et al., 2010a, Cormie et al., 2007c).

4.2.6 Speed testing

Timing gates (Newtest power timing system, Finland) were set up at 0, 10, and 20 m positions to measure 10 and 20 m sprint times. Participants began each sprint from a standard two point start with front foot positioned on a line 30 cm behind the first set of timing gates. Each timing gate was set to a height of 80 cm (approximately hip height). Each participant completed four sprints with at least four minutes separating each trial, with the best time used for analysis.

4.2.7 Body composition testing

Percentage body fat was assessed using a 7-site skinfold procedure. The sites used were the pectoral, triceps, midaxillary, subscapular, anterior thigh, abdominal, and suprailiac skinfolds using the methodology of (Duthie et al., 2006). All skinfold measures at each of the four testing time points were performed by the same experienced practitioner. Body mass was measured using the same electronic scales (Seca gmbh, Germany) accurate to 10 g.

4.2.8 Hormone testing

Saliva samples (approximately 2 mL) were collected by passive drool into a sterile container and stored at -20°C until assay. Saliva samples were collected pre and post training block one and two to assess changes in baseline hormones, as well as pre exercise session (i.e. before each warm up) and post exercise session (i.e. immediately after each training session) for both the complex and strength interventions to assess acute hormone responses to exercise. Saliva

samples were analysed at a later date to determine testosterone and cortisol concentrations by routine ELISA methods (Salimetrics, State College, PA) using a spectrophotometric plate reader (Anthos HTIII, Anthos Labtec International). On the day of analysis, samples were thawed and mixed using a vortex. For testosterone, the lower limit of sensitivity was < 1.0 pg/ml, intra-assay coefficient of variation was between 2.5 and 6.7%, and inter-assay coefficient of variation was between 5.6 and 14.1%. For cortisol, the lower limit of sensitivity was < 0.003 μg/dL, intra-assay coefficient of variation between 3.4 and 3.7%, and inter-assay coefficient of variation between 3.8 and 6.4%.

4.2.9 Statistical analysis

A 2-way mixed model analysis of variance (ANOVA) with repeated measures was used to evaluate training intervention, time and interaction effects for the performance, body composition and hormonal variables. A paired *t*-test with a Bonferonni correction was used for post hoc analysis to determine significant intervention differences in the performance, body composition and hormonal variables post training period. To identify the magnitude of change in performance response between the complex and strength interventions, effect sizes (ES) were calculated using the method and thresholds of Cohen (1988). Pearson's Product Moment Correlation Coefficients (*r*) quantified any associations between the absolute change in each performance measure (post – pre training) and baseline changes in hormones (post – pre training) over the combined 10 week training period. The significance level for all analyses was set at $p \leq 0.05$.

4.3 Results

A significant intervention × time effect was identified in measures of power. Post hoc analysis reported a significant change in bench throw maximum mean power from pre- to post-training in response to the complex intervention (+14.1 ± 14%, $p < 0.01$). No other significant differences in performance were observed pre- to post-training for either the complex or strength intervention (Table 4.3). Larger positive effects were demonstrated for the complex intervention for pre to post training measures in bench throw mean power (ES = 0.92) and jump squat mean power (ES = 0.66), whereas these effects were smaller for the strength intervention (bench throw mean power, ES = 0.05; jump squat mean power, ES = 0.25). Neither the complex nor strength intervention resulted in improvements in bench press, back squat, speed, body fat nor body mass (Table 4.3). A significant time effect was observed over the 10-week training period. Post hoc analysis revealed a significant 2% increase in 1RM bench press strength ($p < 0.01$; ES = 0.30) and a 7% increase in back squat strength ($p < 0.05$; ES = 0.50) (Table 4.4). There was also a 13% increase in bench throw power ($p < 0.01$; ES = 0.80) and a 9% increase in jump squat power ($p < 0.05$; ES = 0.80). There were no significant

changes in body fat from pre- to post-training, but there was a 1% increase in body mass ($p < 0.05$; ES = 0.10). No significant changes in 10 or 20-m sprint times were reported from pre- to post-training. A preferential effect was observed in the participants who performed the complex protocol first followed by the strength protocol (Figure 4.2). These participants (n = 5) demonstrated larger increases in bench throw (ES = 0.68) and jump squat power (ES = 0.88) as well as very large increases in back squat (ES = 1.18) and bench press strength (ES = 1.60; $p < 0.01$) when compared with the participants who performed the strength protocol first followed by the complex protocol (n = 6).

Table 4.3 Performance responses (mean ± SD) to the *complex* training *strength* training interventions pre and post training. ES = effect size.

Assessment	Complex				Strength			
	Pre-training	Post-training	% Change	ES	Pre-training	Post-training	% Change	ES
Bench press (kg)	117.4 ± 11.8	119.3 ± 11.8	1.6	0.16	122.8 ± 20	125.4 ± 22.5	2.1	0.12
Back Squat (kg)	164.2 ± 20.5	167.4 ± 20.3	1.9	0.15	166.0 ± 14.9	175.9 ± 21.9	5.6	0.47
Bench throw (w)	805.5 ± 145.4	937.6 ± 140.9**	14.1	0.92	874.8 ± 163.9	867.2 ± 167.0	-0.9	0.05
Jump squat (w)	4650.2 ± 535.2	5050.0 ± 140.9	7.9	0.66	4673.9 ± 163.9	4849.3 ± 167.0	3.6	0.25
0-10m speed (s)	1.95 ± 0.07	1.97 ± 0.12	1.0	0.20	1.96 ± 0.07	1.99 ± 0.07	1.5	0.43
0-20m speed (s)	3.25 ± 0.10	3.30 ± 0.11	1.5	0.47	3.27 ± 0.10	3.31 ± 0.10	1.2	0.40
Body fat (%)	12.9 ± 3.6	12.9 ± 3.5	-0.2	0.00	13.1 ± 3.2	12.8 ± 3.3	-1.6	0.09
Body mass (kg)	99.5 ± 9.2	100.6 ± 9.5	1.1	0.12	99.9 ± 9.0	100.1 ± 9.3	0.2	0.02

Significantly different from pre-training **$p < 0.01$.

Table 4.4 Pre and post 10 week training changes in performance. ES = effect size.

	Pre-training	Post-training	% Change	ES
Bench press (kg)	117.7 ± 12.0	120.6 ± 12.5*	2.4	0.26
Back Squat (kg)	165.8 ± 16.0	176.9 ± 24.5*	6.7	0.54
Bench throw (w)	840.0 ± 136.1	949.5 ± 143.8**	13.0	0.78
Jump squat (w)	4662.1 ± 537.7	5087.6 ± 530.4*	9.1	0.80
0-10m speed (s)	1.96 ± 0.06	1.99 ± 0.08	1.8	0.42
0-20m speed (s)	3.26 ± 0.10	3.28 ± 0.11	0.6	0.19
Body mass (kg)	99.7 ± 9.1	100.6 ± 9.8*	0.9	0.10
Body fat (%)	13.0 ± 3.4	13.0 ± 3.5	0.0	0.00
Significantly different from pre-training *p<0.05 **p<0.01				

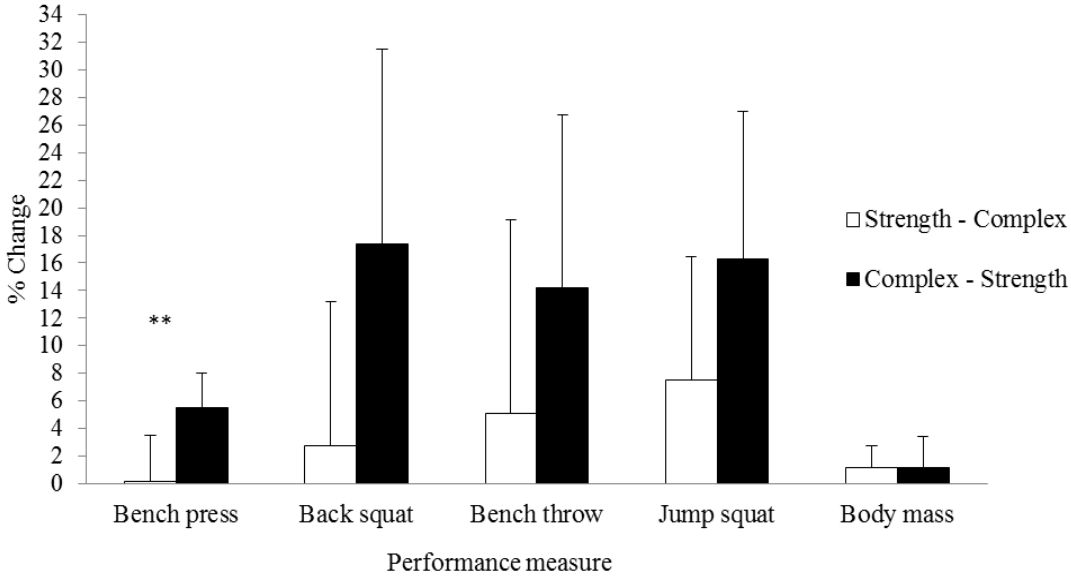


Figure 4.2 Training intervention order effect on performance. Complex-strength significantly different from strength-complex ** $p < 0.01$.

A significant decrease in cortisol ($p < 0.01$) and an increase in testosterone to cortisol ratio ($p < 0.01$) over the experimental period were observed for the acute mean change in the pre to post exercise hormone samples. No significant training intervention or intervention over time effects were realised in any of the acute mean pre- to post exercise changes in hormone samples (Table 4.5) with substantial variability in individual response patterns witnessed over the eight exercise sessions for both complex and strength interventions (Figure 4.3).

When baseline hormones were analysed (pre- to post training), no significant change in testosterone, cortisol or testosterone to cortisol ratio was identified in response to the individual complex and strength interventions (Table 4.6). Similarly, when analysed over the combined 10- week training period, no significant changes in baseline testosterone, cortisol or testosterone to cortisol ratio were identified (Table 4.7). No significant correlations were identified between the acute pre to post exercise change in hormones and changes in any of the performance measures. Significant negative correlations were identified between the pre-post training changes in baseline cortisol and back squat strength (r = -0.69, $p < 0.05$) and baseline cortsiol and jump squat power (r = -0.89, $p < 0.01$), whilst a positive correlation was identified in baseline changes in testosterone to cortisol ratio and jump squat power (r = 0.70, $p < 0.05$) in response to the combined (10-week) training programme. No other significant correlations between changes in baseline hormones and performance were identified.

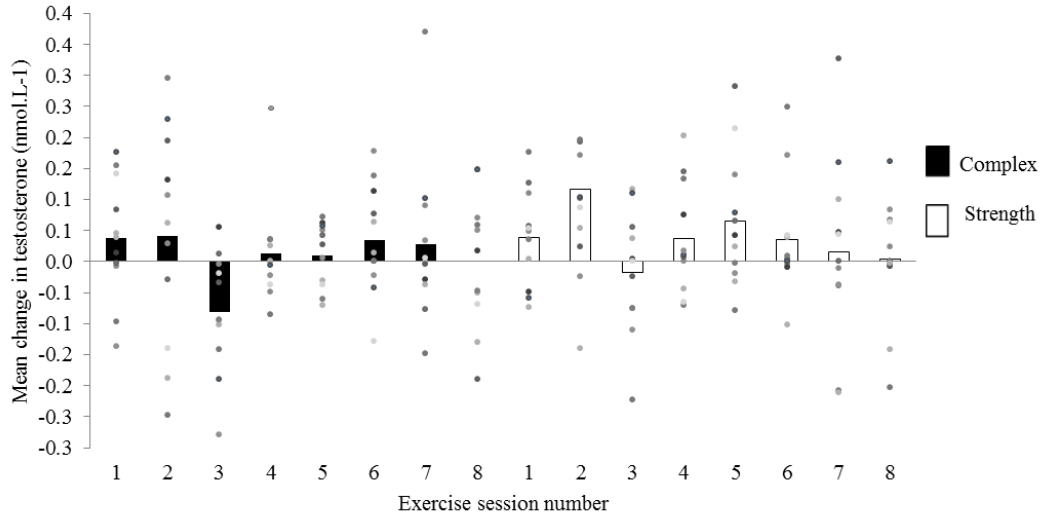
Table 4.5 Mean of all pre- and post exercise session hormone samples for both the *complex* training and *strength* training interventions.

	Complex			Strength		
	Pre-training	Post-training	% Change	Pre-training	Post-training	% Change
Testosterone (nmol.L1)	0.37 ± 0.12	0.38 ± 0.13	3	0.37 ± 0.10	0.41 ± 0.08	11
Cortisol (nmol.L1)	4.38 ± 1.63	3.24 ± 1.49	-26	4.64 ± 1.55	4.12 ± 2.11	-11
T/C ratio (arbitrary units)	0.09 ± 0.02	0.13 ± 0.05	44	0.09 ± 0.03	0.12 ± 0.04	33

Table 4.6 Baseline hormone responses (mean ± SD) to the *complex* training *strength* training interventions pre and post training.

	Complex			Strength		
	Pre-training	Post-training	% Change	Pre-training	Post-training	% Change
Testosterone (nmol.L1)	0.46 ± 0.21	0.37 ± 0.14	-20	0.40 ± 0.12	0.40 ± 0.15	-1
Cortisol (nmol.L1)	6.71 ± 4.29	4.74 ± 1.88	-29	4.74 ± 2.82	4.34 ± 2.15	-8
T/C ratio (arbitrary units)	0.08 ± 0.05	0.08 ± 0.03	3	0.13 ± 0.11	0.11 ± 0.04	-20

A)



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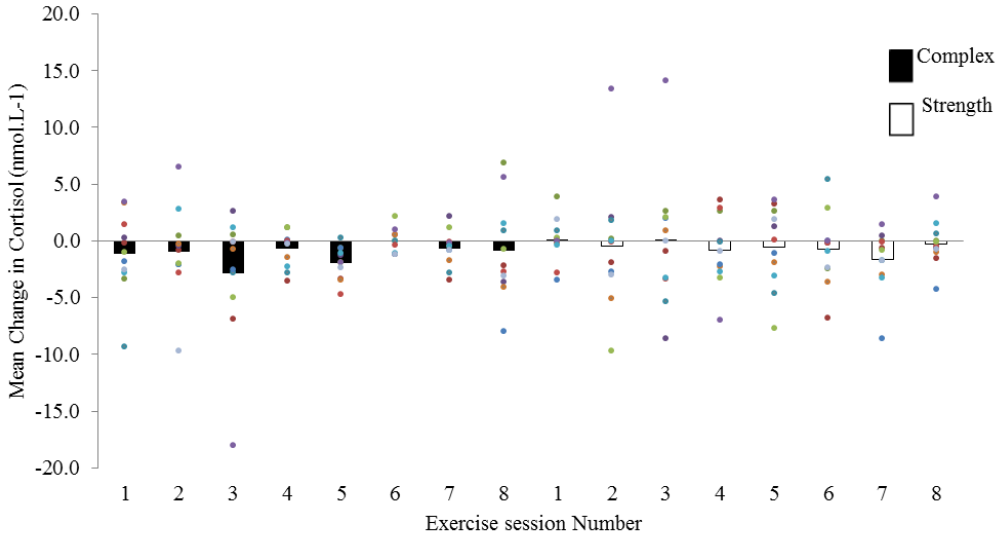


Figure 4.3 Change in hormone measure for the mean group response (bars) and individual response (markers) at each exercise session for the complex training and strength training interventions. (A) Mean change in testosterone, (B) mean change in cortisol.

Table 4.7 Pre and post 10 week training changes in baseline hormones.

	Pre-training	Post-training	% Change
Testosterone (nmol.L1)	0.40 ± 0.13	0.41 ± 0.20	0
Cortisol (nmol.L1)	5.05 ± 2.37	4.84 ± 1.96	-4
T/C ratio (arbitrary units)	0.12 ± 0.10	0.09 ± 0.03	-24

4.4 Discussion

This study investigated the effects of a complex training programme on measures of muscular strength, physical performance, body composition and hormone levels in elite rugby players who were training and playing concurrently during an in-season period. The main findings of this study were that: 1) the complex intervention resulted in greater improvements in measures of power whilst maintaining measures of strength at around baseline values; 2) the combined complex and strength cycle (10 weeks) was effective at initiating significant increases in strength, power and body mass; 3) no significant effects of training were identified for any of the hormone measurements with limited associations between strength and power changes and alterations in baseline hormone concentrations.

This study provides evidence to suggest that executing a repeated complex programme can improve upper and lower limb power characteristics whilst maintaining upper and lower limb strength during in-season training periods. These findings are in agreement with previous research which has suggested that training with a combination of strength and power resistance exercises can result in neuromuscular adaptations encompassing a greater spectrum of the force-velocity curve (Harris et al., 2000, Cormie et al., 2007b). The present findings are similar to those reported by Dobbs et al. (2015) who demonstrated meaningful effect size improvements in measures of vertical and horizontal counter movement jump measures of force (ES = 0.40-0.46) and velocity (ES = 0.62-0.84) after seven weeks of performing complex set exercises. The present study demonstrates that improvements in dynamic performance appear to manifest after four weeks of training, making complex training an effective format to prescribe during an in-season training phase where power development is the main performance outcome.

The preferential change in power after the complex protocol is likely to reflect the specificity of training exercise inherent to both the complex and strength protocols. It must therefore be acknowledged that the differences observed in strength and power may not solely reflect the effects of complex training. However, previous training studies which have utilised a combination of complex multi joint strength and power activities over four to eight week training periods have reported improvements in power to be due primarily to enhanced neural capacity, that is, changes in motor unit recruitment and motor unit firing frequency (Cormie et al., 2007b, McBride et al., 2002). Other neural adaptations, such as reduced intracortical inhibition and enhanced motor coordination, have also been identified in response to short (four-week) multi joint strength training protocols (Weier et al., 2012). The strength training background of the current athletes may also support the concept that neural alterations supported power adaptations in response to the complex intervention. Previously strength trained athletes are reported to have greater potential for faster adaptive improvements in high

velocity movements when exposed to a power training stimulus due to a greater potential to enhance neural drive (Cormie et al., 2010b). For the current athlete population (young elite strength trained rugby players), strength training in isolation over a short four-week period was not sufficient to facilitate alterations in power. However, the combination of force and velocity specific exercises was effective at maintaining strength qualities whilst improving some markers of power.

The complex protocol, which incorporated the principles of PAP, may have also influenced central and peripheral nervous system excitability (Tillin and Bishop, 2009). The potentially elevated neuromuscular state associated with PAP may acutely influence the recruitment and synchronisation of higher order motor units (Gullich and Schmidtbleicher, 1996) as well as increasing phosphorylation of myosin regulatory light chains to enhance actin-myosin cross-bridging cycling rates (Hamada et al., 2000b). Consequently, over repeated complex exposures, these short-term nervous system responses may have accumulated to support the neuromuscular adaptations associated with the improvements in power (Docherty D, 2004, Comyns et al., 2010). It is clear that the design of the complex intervention of the present study supported the development of power in measures of bench throw and jump squat performance. The mechanistic effects of PAP are however unclear in the present context as the current study did not include assessment of muscle potentiation (e.g. measures of muscle twitch response or H-reflex amplitude).

When performance was analysed as a whole over 10 weeks, the cumulative effects of a complex and strength training intervention resulted in significant gains in all strength and power variables. Previous research into the effects of short-term isolated strength, power and endurance training has reported similar improvements to this study irrespective of the potential interference effects associated with concurrent training (Hakkinen et al., 2002a, McCarthy et al., 2002), particularly in relation to elite team sport athletes during pre and in-season training periods (Appleby et al., 2012, Comfort et al., 2012). Both protocols were therefore effective at improving performance when combined with the applied training and playing loads of a professional rugby union team. The complex and strength training protocols were, however, not effective at improving 0-10 m or 0-20 m speed times. This finding is in agreement with previous strength and power training studies (Harris et al., 2000, McBride et al., 2002), and may reflect the lack of specific speed training in either training programme.

Despite all participants being exposed to similar training volumes, the participants who performed the complex protocol first made greater gains than those who performed this after the strength programme (Figure 4.2). It has been suggested that optimal adaptation to a specific training intervention depends on the appropriate selection of an overload stimulus based on the neuromuscular characteristics most susceptible to change (Cormie et al., 2011c).

With this in mind, each participant in the present study performed six weeks of baseline strength training prior to the current intervention period. It may be theorised that the current athletic population may therefore have been more receptive to a training programme which incorporated both strength and power components. These findings may highlight the importance of effective programme design for attenuating the negative effects of diminished returns in athletes with prior training experience. The complex-strength order athletes may have experienced more appropriate training prescription which widened the potential for neuromuscular adaptation over the 10 week training period when compared to the strength-complex order athletes.

The complex and strength exercise protocols demonstrated no significant intervention effects on acute alterations in hormones from pre to post exercise. At an individual level, large variability in pre to post exercise changes in testosterone and cortisol concentrations were identified over the course of the eight exercise sessions for both the complex and strength interventions (Figure 4.3). Individualised protocol-specific hormonal responses have been identified previously in acute athletic studies (Beaven et al., 2008b). Subsequently, it has been suggested that resistance exercise protocol prescription could be selected based on an individual's "testosterone responsiveness" to ensure training elicits optimal anabolic and adaptive outcomes (Beaven et al., 2008a). However, the session to session within individual variability witnessed in the current study suggests a lack of reproducibility in the hormone response to the same exercise stimulus in these elite performers.

The variability observed in the acute exercise and baseline hormonal response patterns are not unusual for athletes in an applied environment. Previous research into acute and baseline changes in hormones for elite team sport athletes training concurrently during competition periods have reported fluctuations in testosterone and cortisol similar to the patterns observed in the present study (Argus et al., 2009, Cormack et al., 2008, Crewther et al., 2013, Kraemer et al., 2004). The reported endocrine variation may be related to modifications in the action of the hypothalamo-pituitary-adrenal axis in response to the varied physiological load initiated through weekly training, competition and recovery stimuli that characterise applied sporting environments (Cormack et al., 2008). Alternatively, such variability may simply reflect biological variation which may limit the role of hormone monitoring as biomarkers for selecting optimal training strategies in the applied athletic environment.

When acute pre to post exercise hormone response patterns were analysed over the combined training period, there was some evidence to suggest training initiated reductions in cortisol and an increase testosterone to cortisol ratio. However, in contrast to the original hypothesis no correlations were identified between pre to post-acute exercise changes in hormones and combined training performance responses. Repeated manipulation of the hormonal

environment immediately post exercise was not therefore required for chronic changes in performance to be realised over the 10 week training period. Similarly, most of the changes in strength, power and body composition as a consequence of the combined training programme were unrelated to changes in baseline hormones. These findings are in agreement with evidence indicating testosterone availability in response to resistance training programmes are not necessary for the adaptive mechanisms associated with improved muscle strength and anabolism to be initiated (Mitchell et al., 2013, West et al., 2009)

4.4.1 Limitations

It must be recognised that any mechanisms driving the observed improvements in power and strength can only be theorised since alterations in muscle potentiation and nervous system adaptation were not monitored and so this is a limitation of the present analysis. Also, the short training duration may present only a "snapshot" of the neuromuscular, hormonal and performance adaptation which can be potentially realised in response to both training interventions. However, the current study managed to identify the efficacy of complex training in the applied setting and the current time frames of the training cycles are a representation of a realistic strength and power cycle which elite team sport athletes are exposed to during in-season periods. Future training studies into the effectiveness of complex training may look to analyse the mechanisms associated with neuromuscular enhancement over extended training periods to fully elucidate the adaptations associated with this modality of training.

Whilst the use of saliva collection for subsequent analysis of testosterone and cortisol in the current applied setting is practical, it must also be recognised that this environment may have induced measurement error. It has been demonstrated that prolonged storage (i.e. > 28 days) of saliva could reduce the concentrations of testosterone available for analysis and therefore reduce the reliability of analysis in this measure (Toone et al., 2013). The current study stored the saliva samples under freezer conditions (i.e. ~ -20º) which are reported to be sufficient to preserve the original concentration of hormone (Toone et al., 2013). However, the storage duration was longer than 28 days and this should be a consideration when interpreting the hormone results in the present study

The two week recovery period used to separate the interventions can also be considered relatively short. This may have influenced the observed strength and power outcomes associated with the two training interventions. However, this time frame was chosen to ensure assessments of training effectiveness could be made during in-season periods. Whilst a four week recovery period could have been employed during the off-season, the training interventions would have been performed during two different phases of the season (i.e. end of season and pre-season). This would have reduced the standardisation of external training loads

and limited the suggested application of complex training as an effective in-season training method. Finally, the two minute recovery intervals prescribed during the strength training intervention may not have been of appropriate duration to allow for sufficient recovery between exercises (i.e. reduced phosphocreatine replenishment potential). This may have negatively influenced the mechanical intensity that each athlete could have generated during the repeated set design of the strength intervention.

4.5 Conclusions

The use of a complex training protocol over a four week period was effective at enhancing selected power measures whilst maintaining strength characteristics in young elite rugby players during a mid-season training phase. This mode of training appears to facilitate the transfer of force production to power expression in previously trained young athletes. The combination of complex and strength cycles performed twice weekly over a 10 week concurrent training and playing period also proved to be an effective strategy for promoting positive adaptations in strength, power and body mass. Finally, the variability in endocrine responses to both acute exercise and pre to post training baseline hormones suggests improvements in performance capacity were achieved irrespective of systematic circulating hormonal factors. The present study demonstrates the unpredictability of testosterone and cortisol monitoring as markers of acute workout and chronic training efficacy in an applied concurrent athletic environment.

4.6 Practical applications

Complex training is a useful training modality for athletes who require strength maintenance and power development during time-restricted concurrent training periods. This training modality may be suited to in-season training periods when an effective method for increasing power is required but training frequency is limited. It is recommended that strength exercise (83-92% 1RM) coupled with power (40-60% 1RM) and plyometric movements be programmed to elicit meaningful improvements in bench throw and jump squat power. Utilising a complex training cycle in between focused strength training cycles may also provide the training variation and progression required for a wider spectrum of force and velocity adaptation to be realised in elite rugby athletes.

CHAPTER 5: SHORT-TERM EFFECTS OF A WEIGHTLIFTING AND CYCLE SPRINT PRE-CONDITIONING PROTOCOL ON HORMONAL RESPONSES AND POWER DEVELOPMENT

5.1 Introduction

Post-activation potential (PAP) is a widely accepted phenomenon that describes the positive effects of a high intensity pre-conditioning exercise on subsequent dynamic motor performance. The classic proposed mechanisms of PAP include an increase in actin and myosin sensitivity to Ca^{2+} through the phopshorylation of myosin regulatory light chain (Docherty D, 2004), as well as an elevation in spinal reflex processing (Güllich, 1996). Together, these physiological effects may increase the rate and recruitment of higher threshold motor units during subsequent dynamic activity (Hodgson et al., 2005). Other mechanisms, such as short-term alterations in endogenous hormones, have been suggested to mediate acute physical performance through non-genomic effects on the neural system, muscle contractile kinetics, as well as behaviour and cognition (Crewther et al., 2011b, Cook and Crewther, 2012). However, manipulation of the hormonal environment to enhance acute performance via pre-conditioning exercise remains incompletely understood.

The potential association between testosterone and neuromuscular function has received interest in athletic based studies. Acute high intensity cycle sprint warm-up strategies have been utilised to initiate rapid elevations in salivary testosterone concentrations which appear to support subsequent workout performance in measures of lower and upper body strength (Crewther et al., 2011b). Cook et al. (2014) also demonstrated that morning weight training in elite rugby players enhanced afternoon performance in measures of speed, strength and power whilst attenuating the circadian decline in testosterone. Whilst current evidence does not implicate a causal relationship for testosterone and physical performance, testosterone may be a marker for predicting physical performance capacity in elite athlete populations (Cardinale and Stone, 2006, Crewther et al., 2012b). Of particular interest to PAP research is that if circulating hormones have a role in neuromuscular enhancement, then pre-conditioning exercise in one muscle group could provide systemic neuromuscular enhancement for other muscle groups as long as circulating testosterone concentrations are increased. Whilst Chapter

4 reported equivocal findings for the chronic role of hormones in support of training adaptation, assessing the effects of steroid hormones on acute systemic muscle potentiation could therefore be of practical relevance for the prescription of applied training strategies.

Pre-conditioning contraction type and intensity are two of the key factors responsible for the development of PAP (Tillin and Bishop, 2009). Consequently, investigating the efficacy of different pre-conditioning contraction modes on dynamic performance and hormones in an applied training setting warrants further investigation. In particular, high intensity lower limb weightlifting and cycle sprint exercise are two commonly utilised training strategies which, in isolation, have both been associated with acute alterations in endogenous testosterone (Raastad et al., 2001, Beaven et al., 2008b, Crewther et al., 2011b). These strategies have also been utilised as a means of potentiating dynamic performance and both traditional and hormonal mechanisms have been suggested as rationale for the observed elevations in neuromuscular function (Obminski et al., 1998, Chiu et al., 2003, Gourgoulis et al., 2003, Duthie et al., 2002, Crewther et al., 2011b).

The primary aim of this study was to compare the potentiating effects of a cycle sprint and weightlifting exercise intervention on subsequent measures of power in both the lower and upper limb musculature of elite rugby players in order to assess the influence of pre-conditioning exercise mode and local versus systemic effects. The secondary aim was to analyse the acute effect of both interventions on blood concentrations of free-testosterone and cortisol, and determine any association between exercise induced alterations in hormones and lower and or upper body power. Whilst performance and hormonal effects were analysed for both protocols, the cycle sprint was designed to induce hormonal priming whilst the leg press was designed to induce potentiation. It was hypothesised that both pre-conditioning exercise interventions would increase power in an applied elite rugby environment, and improvements in power would be associated with systemic alterations in the hormonal milieu.

5.2 Materials and methods

5.2.1 Experimental design

A randomised, test-retest design was used to assess the effects of three experimental conditions on upper and lower body measures of force and power in elite rugby players over a four week period. The three exercise conditions comprised: a) a weightlifting protocol, b) a cycle sprint protocol, and c) a control (no prior loading) protocol (Figure 5.1). The design allowed for

comparison (i.e.test-retest) of the effects of contractile history between normal dynamic performance (control) and the dynamic performance achieved following a weightlifting or cycle sprint potentiation stimulus. Peak power output, rate of force development and peak force were chosen as the primary outcome measures because all three are important physical attributes for elite rugby players (Deutsch et al., 2007). In the week before the first main trial, participants performed a familiarisation session which included determination of each participant's 1RM leg press; in addition, participants were familiarised with the cycle sprint and dynamic performance measures. Each participant was then randomly allocated to one of the three protocols via the use of a random number generator (Microsoft Office Excel). One week separated each testing protocol; each protocol was performed at a similar time of day (9 AM ± 1 hour) to account for diurnal variation (Kraemer et al., 2001).

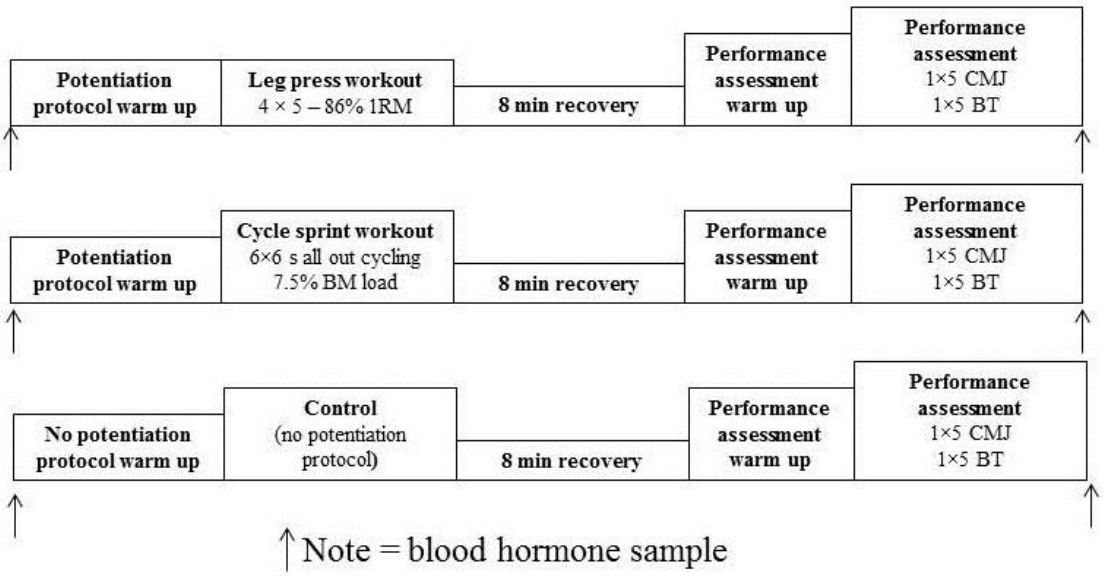


Figure 5.1 Schematic representation of the experimental potentiation and control workouts. CMJ = counter movement jump, BT = bench throw, RM = repetitions maximum, BM = body mass.

5.2.2 Participants

Fourteen professional male rugby players (mean ± SD: age 20 ± 1 years, mass 97 ± 10 kg, height 186 ± 5 cm, and sum of eight skinfolds 93 ± 21 mm) with at least two years strength and power training experience participated in this study. Testing procedures were carried out during the mid-season phase of a national rugby competition. During the experimental period participants were performing intensive but low volume training external to that prescribed in

the current study which included; power and strength, metabolic conditioning and technical and skills based training. They also played in two official games. Each participant had the risks and benefits of the experiment explained to them and provided written informed consent. Study procedures complied with the regulations of the Declaration of Helsinki (2008) and were approved by an institutional ethics committee (Research Ethics Approval Committee for Health at the University of Bath).

5.2.3 Potentiation protocols

Weightlifting potentiation protocol

The weightlifting protocol was performed on a bilateral leg press machine (A-Unit Leg Press, A-Unit Ltd, Derby, UK). The leg press exercise has been shown in previous exercise studies to be an effective and practical tool for stimulating acute elevations in the hormonal environment (Ahtiainen et al., 2003, Beaven et al., 2008b). Equally, the prescription of high intensity multi-joint strength exercises performed with load equating to 80-90% 1RM performed for 3-6 repetitions for multiple sets has been demonstrated to be an effective stimulus to significantly elevate acute testosterone (Raastad et al., 2000, Beaven et al., 2008b, Beaven et al., 2011). Prior to the leg press protocol each participant was prescribed a load which corresponded to 86% of their 1RM via a prediction equation (Beachle, 2000). The leg press protocol consisted of a progressive warm up (1 × 6 repetitions at 60% 1RM, 1 × 4 repetitions at 70% 1RM, 1 × 2 repetitions at 80% 1RM), followed by 4 × 5 repetitions at 86% 1RM with a three minute rest period between sets. The volume and intensity were selected to provide sufficient stimulus to initiate the mechanisms associated with PAP, as used previously (Kilduff et al., 2008, Comyns et al., 2010). Resistance exercise studies utilising loads at and above 85% 1RM for repeated sets have also been shown to elicit significant acute elevations in the hormonal environment in strength trained athletes (Kraemer et al., 1990, Beaven et al., 2008b).

Cycle sprint potentiation protocol

The cycle sprint protocol was performed on a watt bike (Wattbike Pro, Wattbike Ltd, Nottingham, UK). Seat height was adjusted for each individual and participants remained seated throughout the protocol. The cycle sprint protocol consisted of a brief warm up (5 min continuous cycle at 40 w), followed immediately by a repeated sprint exercise bout consisting of 6 × 6 s all-out efforts separated by 24 s of rest (Girard et al., 2011, Bishop and Edge, 2006). A constant load corresponding to 7.5% of each participant's body mass was applied to the watt

bike (French et al., 2003). A high intensity all-out cycle sprint protocol was chosen as this exercise has been shown previously to acutely elevate free-testosterone concentrations and potentiate neuromuscular performance in elite male rugby players (Crewther et al., 2011b).

Both potentiation protocols were chosen based on their availability and ease of use within the applied training setting. After both potentiation protocols, participants rested for eight minutes before completing the dynamic performance assessment (Figure 5.1). Eight minutes of recovery was chosen as this time period is of sufficient duration to allow elite rugby players to develop muscle potentiation and enhance power (Kilduff et al., 2008). Prior to each performance assessment participants performed a brief warm up which incorporated progressive intensity body mass counter movement jumping combined with bench throws (1×4 repetition jumps at 80% relative intensity, 1×4 repetition bench throw at 20% group 1RM, 1×2 repetition jumps at 90% relative intensity, 1×2 repetition bench throw at 30% group 1RM).

Control protocol

No potentiation stimulus was employed for the control protocol. Upon arrival into the testing facility participants rested for eight minutes. Following the initial rest period, each participant performed the pre- performance assessment warm up. After the warm up, each participant performed the dynamic performance assessment (Figure 5.1).

5.2.4 Dynamic performance assessment

Bench throw

Upper body power was assessed via bench throws performed on a smith machine using controlled eccentric and ballistic concentric movements. Concentric peak power output was calculated for the bench throws using an optical encoder analysis system (Gymaware, Kinetic Performance Technology, Canberra, Australia) which has been reported to provide valid measures of kinetics during bench throw movements (Drinkwater et al., 2007). At each assessment, five repetitions were performed to ensure the highest instantaneous power outputs were identified (Baker and Newton, 2005) with the mean of the five values used for further analysis. Testing for the bench throws was assessed with a resistance of 54 kg which represented an average of 40 ± 4% of the group's 1RM bench press. This absolute resistance was chosen as 40% 1RM has been shown to be optimal for developing peak power output in the upper body for elite male rugby players (Kilduff et al., 2007). An absolute load for power testing has been utilised previously (Baker and Newton, 2007) and has been shown to be more

sensitive to intervention-induced changes in power when compared with the use of a relative resistance. This method also exhibits less inter-individual variation compared with using a designated percent 1 RM for each individual (Baker and Newton, 2007). Analysis of test-retest reliability was conducted with data from the five consecutive bench throw trials performed during the control. For measures of peak power the ICC was 0.96, and the CV was 2.6%.

Counter movement jump

Lower body peak force and rate of force development were assessed via a body mass counter movement jump performed on a portable force platform (AMTI ACCUGAIT 0341, USA) operating at a sample rate of 200 Hz. Participants were instructed to descend to a self-selected depth followed by a maximal jump for height. To exclude the influence of arm swing on jump height (Harman et al., 1990), each participant was instructed to keep his arms at his side during each jump. At each assessment, participants performed five counter movement jumps with the mean of the five values used for further analysis. A body mass load for each participant was selected as this has previously been shown to provide the optimal resistance for the expression of lower body peak power in elite rugby players (Bevan et al., 2010a). Analysis of test-retest reliability was conducted with data from the five consecutive counter movement jumps performed during the control. For measures of peak force the ICC was 0.96, and the CV was 2.9%, for measures of rate of force development the ICC was 0.96 and the CV was 14.5%.

5.2.5 Data processing

The vertical component of the ground reaction force during the counter movement jump was used for performance analysis. Force data collected via the AMTI portable force platform was exported to Microsoft Office Excel. Peak force was identified as the value closest to 5 N below the highest absolute force value recorded. Force onset was identified as the point at which ground reaction force took on a positive gradient (inclusive of eccentric and concentric muscle force production), as the value closest to 5 N above the lowest ground reaction force time point. Force termination was identified as the peak force value. The rate of force development was identified by calculating the gradient of the linear least squares fit through the positive force production slope i.e. force onset value to force termination (Figure 8.2).

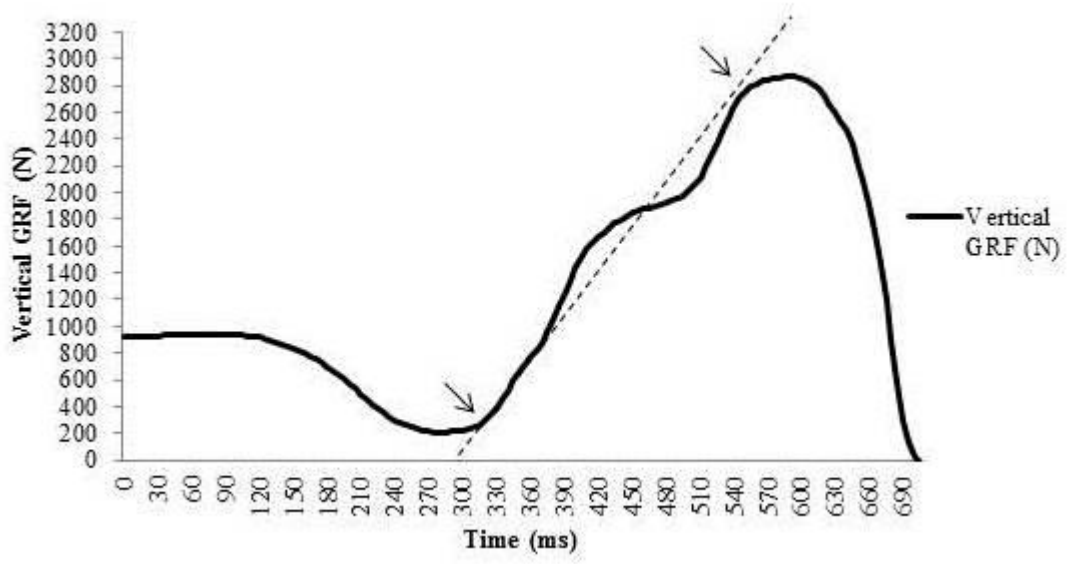


Figure 5.2 Example force-time trace for the counter movement jump, with arrows identifying the approximate point of onset and termination of positive force production. Dotted line represents the slope of positive force production (linear least squares fit) from which rate of force development was calculated. GRF = ground reaction force.

5.2.6 Hormone analysis

Hormone concentrations were measured using capillary blood samples for determining plasma free testosterone and total cortisol. Capillary blood samples (500 μL) from a fingertip were collected and stored on ice in ethylenediaminetetraacetic acid prepared collection tubes (Microvette 500, Sarstedt, Numbrecht, Germany). For the cycle sprint and leg press protocols, samples were collected immediately prior to the potentiation protocol warm up (pre), and directly after (post) the performance assessment (Figure 5.1). For the control protocol, samples were collected upon immediate arrival into the testing facility (pre) and directly after (post) the performance assessment (Figure 5.1). After collection, each sample was centrifuged at 10,000 rev/min for 5 minutes. Plasma was then dispensed into a microtube and stored at -20˚C until further analysis. On the day of analysis, samples were thawed and mixed using a vortex. Plasma was analysed for free-testosterone and cortisol concentrations in duplicate by a competitive ELISA method, using commercially available kits (Free-testosterone enzyme immunoassay kit; Cortisol enzyme immunoassay kit, both IBL International, Germany). The minimum detection limit for the free-testosterone assay was 0.1 pg/mL with intra and inter-assay coefficients of variation (CV) of < 8.90% and < 14.64% respectively. The minimum

detection limit for the cortisol assay was 2.46 ng/mL with intra and inter-assay CV of < 3.47% and < 5.00% respectively. Assay plates were read using a spectrophotometric plate reader (Anthos HTIII, Anthos Labtec International).

5.2.7 Statistical analysis

Statistical analysis was conducted using IBM SPSS Statistics (ver.19). All values were represented as Mean ± SD and statistical significance was taken at $p \leq 0.05$. Performance and hormonal data was analysed as a pooled group. A one-way ANOVA was used to determine if significant differences existed in values of peak force, rate of force development and peak power output for each exercise protocol against the control. A two-way ANOVA was used to determine if significant differences existed between pre to post sample (time) and between protocol values in free-testosterone, cortisol and free-testosterone to cortisol ratio. All the data was tested for sphericity and paired t-tests were used for post hoc analysis. All data sets were assessed for normality through the use of the Shapiro-Wilk test. To identify the magnitude of change in performance and hormone response to the three testing protocols, effect sizes were calculated using the method and thresholds of Cohen (1988). Pearson's Product Moment or Spearmans Rank Correlation Coefficients (r) were implemented to assess if associations existed between changes in measures of performance relative to the control (i.e. peak force and rate of force development) against each participants baseline strength status (leg press 1RM) as well as protocol induced hormone changes (as a percentage relative to the control) against protocol induced performance changes (as a percentage relative to the control).

5.3 Results

The one way ANOVA reported no between protocol differences ($p > 0.05$) in absolute measures of counter movement jump peak force, rate of force development and bench throw peak power output (Table 5.1). Although no significant differences or meaningful effects were observed between protocols at a group level, the cycle sprint and leg press protocols demonstrated heterogeneity in performance responses when compared with the control protocol (Figure 5.3).

Significant positive correlations were observed between baseline strength and the change in peak force and rate of force development relative to the control in response to the cycle sprint protocol (Figure 5.4) No significant correlations were observed between baseline strength and

changes in peak force (r = 0.15, p = 0.32) or rate of force development (r = 0.11, p = 0.38) relative to the control in response to the leg press protocol.

A significant protocol × time interaction effect ($p < 0.01$) was identified for the free-testosterone measure. Post hoc analysis revealed a significant increase in cycle sprint free-testosterone response compared with the control ($p < 0.01$; Table 5.2) and leg press protocols ($p < 0.01$; Table 5.2). A significant main time effect ($p < 0.01$) was identified for pre-post changes in free-testosterone. Post hoc analysis revealed performance of the cycle sprint protocol induced significant elevations in free-testosterone concentration ($p < 0.01$; $d = 0.46$) (Table 5.2). No significant differences in protocol induced changes in cortisol were identified. No significant correlations were identified between the percentage change in free-testosterone and cortisol versus the percentage change in peak force, rate of force development and power in response to either pre-conditioning intervention despite significant elevations in free-testosterone in response to the cycle sprint protocol (Table 5.3). Both the cycle sprint and leg press protocols demonstrated within and between individual variation in measures of free-testosterone and cortisol when compared to the control (Figure 5.5).

Table 5.1 Performance responses to the control, cycle sprint and leg press protocols during countermovement jumps and bench throws (mean ± SD).

Performance measure	Control	Cycle sprint	Cycle sprint % change vs. control	Effect size	Leg press	Leg press % change vs. control	Effect size
CMJ peak force (N)	2424.4 ± 338.9	2393.4 ± 321.4	-0.01	-0.09	2411.3 ± 326.3	-0.01	-0.04
CMJ RFD (N·S)	7151.8 ± 3599.2	6941.8 ± 4320.6	-0.03	-0.05	7131.3 ± 4253.6	0.00	-0.01
Bench throw peak power (W)	1105.4 ± 127.0	1107.4 ± 133.2	0.00	-0.02	1097.4 ± 128.4	-0.01	-0.06

CMJ: Counter movement jump; RFD : Rate of force development

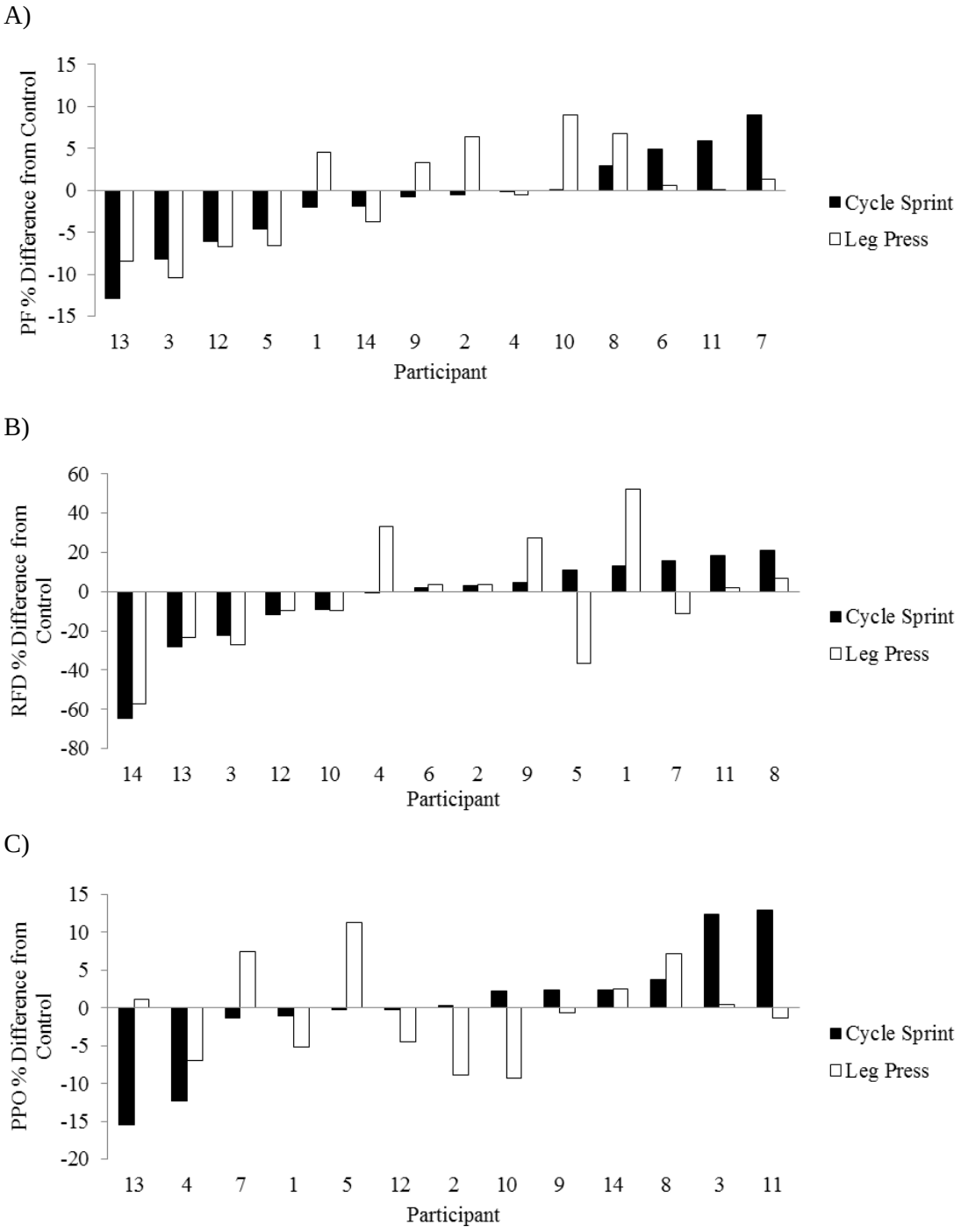


Figure 5.3 Individualised potentiation protocol dependent performance response. Positive values indicate performance increase, negative indicate performance decrease. A) % difference in counter movement peak force (PF) relative to the control, B) % difference in counter movement rate of force development (RFD) relative to the control and C) % difference in bench throw peak power output (PPO) relative to the control.

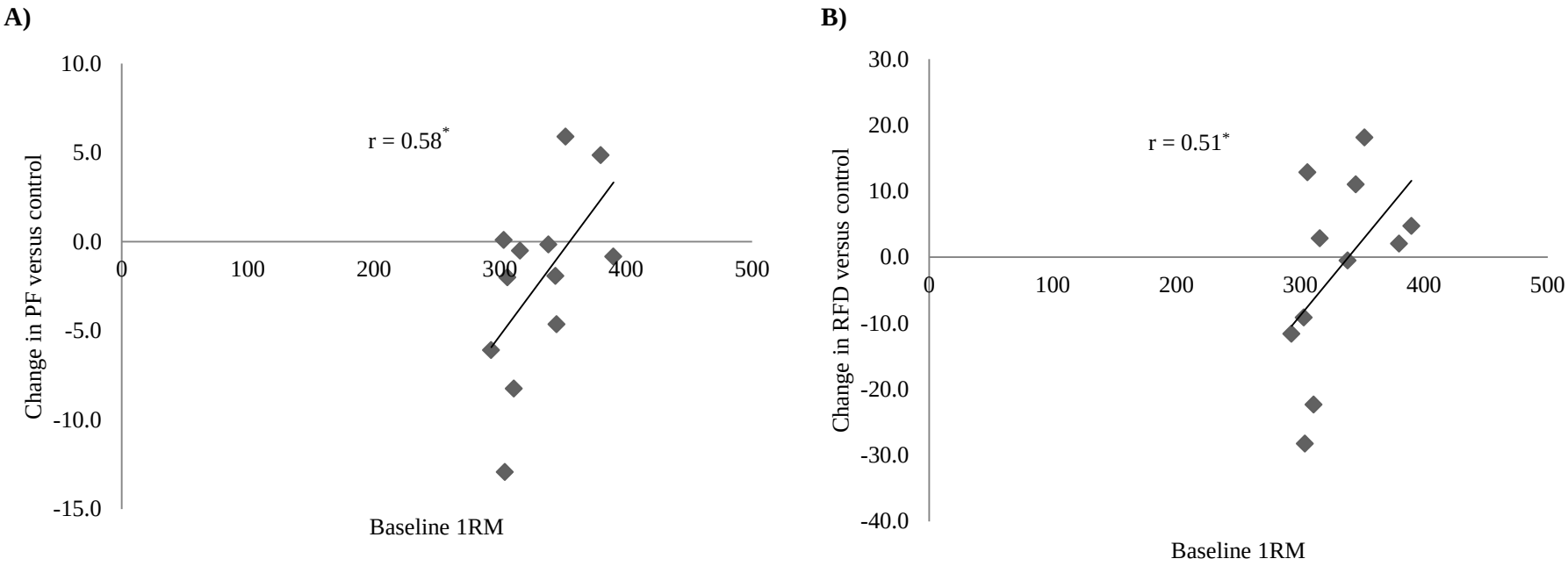


Figure 5.4 Relationship between baseline strength and changes in peak force (A) and rate of force development (B) in response to the cycle sprint protocol. Significant ($p < 0.05$) correlation. PF = peak force, RFD = rate of force development.

Table 5.2 Hormonal responses to the control, cycle sprint and leg press protocols (mean ± SD).

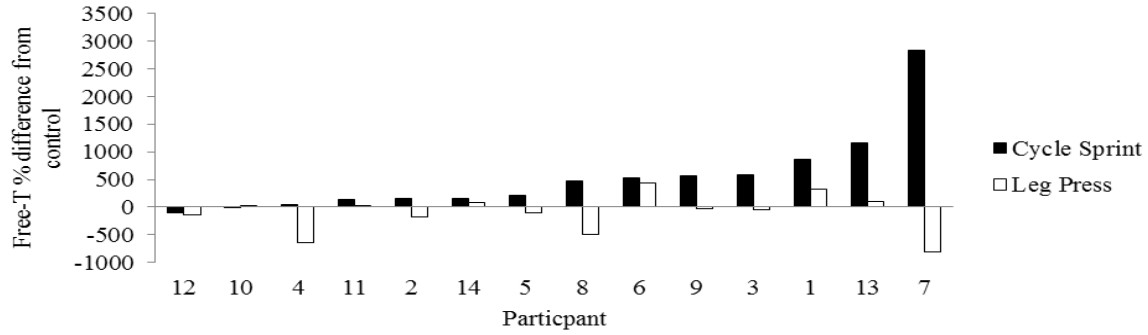
	Control				Cycle sprint				Leg press			
Hormone measure	Pre	Post	% Change	Effect size	Pre	Post	% Change	Effect size	Pre	Post	% Change	Effect size
Free-testosterone (pg/mL)	5.6 ± 2.7	5.5 ± 2.3	-0.8	0.02	5.6 ± 3.1	7.1 ± 3.2**	25#	0.46	5.8 ± 2.4	5.6 ± 2.1	-2.4	0.06
Cortisol (ng/mL)	126.1 ± 40.8	115.2 ± 35.7	-8.6	0.13	120.9 ± 37.0	136.6 ± 60.1	13	0.12	123.7 ± 47.7	118.4 ± 40.1	-4.3	0.12

Table 5.3 Correlations (*r*) with significance value (*p*) between the percentage change in performance measures (relative to the control) against the percentage change in hormones (relative to the control) in response to; A) cycle sprint protocol, B) leg press protocol.

A)	Free-testosterone		Cortisol	
	r	*p*	*r*	*p*
Peak force	-0.11	0.71	0.28	0.33
Rate of force development	0.16	0.58	0.39	0.18
Peak power output	-0.24	0.44	-0.14	0.66

B)	Free-testosterone		Cortisol	
	r	*p*	*r*	*p*
Peak force	-0.11	0.71	-0.05	0.87
Rate of force development	0.01	0.98	0.04	0.90
Peak power output	-0.25	0.41	-0.24	0.43

A)



B)

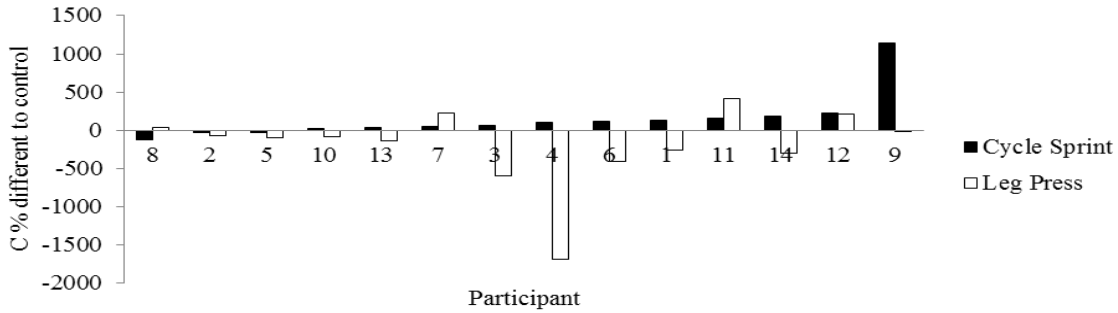


Figure 5.5 Individualised protocol dependent hormone response. A) % difference in free-testosterone (Free-T) relative to the control, B) % difference in cortisol (C) relative to the control.

5.4 Discussion

This study investigated the effects of two pre-conditioning exercise protocols on measures of lower body peak force, rate of force development, upper body power and steroid hormones in a professional rugby population. No overall group improvements in local or systemic performance were realised in response to either protocol. Both pre-conditioning protocols produced individual variations in whole body performance responses, which may to some extent be related to the base strength of individual participants. It appeared that the participants with greater strength levels at baseline may have experienced greater lower body performance responses to the cycle sprint pre-conditioning stimulus whereas participants with lower strength levels at baseline may have experienced detrimental lower body performance responses to the cycle sprint protocol. The cycle sprint protocol proved effective at elevating free-testosterone; however, this response had no positive effects on local or systemic muscle performance. Equally, no other hormone-performance associations were identified which suggests the existence of the non-genomic effects of steroid hormones on neuromuscular performance was not realised within the current acute applied training environment.

The different performance responses between the participants with greater baseline strength to the cycle sprint exercise may in part reflect the strength-potentiation association. This theory suggests that stronger athletes have a greater capacity to overcome fatigue and develop potentiation when compared with weaker athletes (Kilduff et al., 2007, Hamada et al., 2003). Similar to the findings of the present study, Duthie et al. (2002) identified significant relationships between an athlete's baseline measure of strength (1RM) and their capacity to enhance or degrade peak power (r = 0.73) and peak force (r = 0.66). As stronger athletes are characterised by greater myofibrillar cross sectional area of type II muscle fibres (Cormie et al., 2010b), the current findings may reflect the greater capacity that type II muscle fibres possess in developing the neural and mechanical mechanisms associated with PAP (Sweeney et al., 1993, Güllich, 1996). Consequently, the weaker participants at baseline failed to enhance neuromuscular performance potentially due to an inability to overcome the fatigue developed via the cycle sprint protocol (Hamada et al., 2003), whilst the stronger individuals were either unaffected or enhanced performance in response to the cycle sprint pre-conditioning strategy.

The lack of any large meaningful group improvements in performance suggests that the design of the leg press and cycle sprint exercises may not have achieved the correct balance between potentiation and fatigue (Rassier, 2000). This may have been due to the high intensity multiple set and repetition design of both protocols. It has been reported that as the volume of a pre-

conditioning contraction increases so does fatigue and its dominance in the PAP-fatigue relationship (Tillin and Bishop, 2009). The design of the PAP interventions in the current study was partly developed to initiate a hormonal response. Whilst free-testosterone was elevated via the cycle sprints, the high volume nature of both protocols may have limited the group's ability to create a positive net balance of muscle potentiation during the eight minute recovery period. Equally a positive muscle potentiated state in response to both protocols may have been realised at time points not assessed in the current design (e.g. after eight minutes) as inter-subject variability in PAP has previously been attributed to the duration of recovery intervals employed (Kilduff et al., 2008, Bevan et al., 2010b).

The finding that the cycle sprint protocol produced significantly elevated free-testosterone concentrations is supported by previous research (Raastad et al., 2001, Goto et al., 2007, Crewther et al., 2011b). Whilst evidence is sparse, previous studies have demonstrated that acute elevations in anabolic hormones support the performance of power and strength expression (Viru and Viru, 2005, Crewther et al., 2009c). In particular, acute increases in testosterone (9-13%) in response to a cycle sprint exercise demonstrated an association with improvements in leg power 15 minutes after the initial sprint activity (Obminski et al., 1998). However, whilst the cycle sprints produced favourable elevations in the groups anabolic profile (up to 23% in free-testosterone after eight minutes), no hormone-performance potentiation associations in lower or upper body power measures were realised. It appears that both local (lower limb) and systemic (upper limb) muscle performance was unaffected by the increased availability of biologically available testosterone. In contrast to previous evidence (Crewther et al., 2011b, Crewther et al., 2009c) the non-genomic effects of testosterone on neuromuscular performance within a professional rugby union paradigm was not reproduced in this present analysis.

The present study identified between-athlete variability in the hormone response patterns. Support for the inter-individual difference in hormone response to exercise stimuli has been previously documented (Raastad et al., 2000, Hakkinen and Pakarinen, 1993). Whilst free-testosterone was elevated within the group in response to the cycle sprints, free-testosterone and cortisol demonstrated large variability between athletes after the leg press protocol. When hormone analysis has been examined acutely and over repeated training periods it is clear that testosterone and cortisol represent unstable biological markers, potentially due to the varied physiological load that characterise applied sporting environments (Cormack et al., 2008, Argus et al., 2009, Crewther et al., 2013, Mitchell et al., 2013). With the variability and lack of

performance associations observed in the present study, it appears that structuring pre-exercise routines for the purpose of developing an endocrine response may not support universal improvements in training outputs. Equally the cycle sprint exercise may have initiated negative localised performance responses in the participants with lower strength levels at baseline (Figure 5.4). The process of initiating hormonal responses via physical activities could therefore lead to detrimental performance effects in athletic groups who are characterised by a wide range of neuromuscular abilities such as team sport athletes.

5.4.1 Limitations

The limitations of the current study are recognised. Firstly, mechanistic analyses of the central and peripheral muscle factors associated with PAP were not monitored. Consequently, the suggestion that weaker athletes were unable to enhance muscle potentiation due to the maladaptive effects of prolonged fatigue can only be conjectured. Secondly, the present study did not repeat each testing procedure; therefore the individualised performance responses to both protocols could be a reflection of natural variability. Further research is needed to identify if individualised response patterns, particularly between athletes who are categorised as stronger at baseline, are repeatable before the efficacy of PAP protocol prescription can be determined.

5.5 Conclusions

The two pre-conditioning training activities offered no clear benefit on subsequent dynamic performance in the current professional athletic group. However, it appeared a strength potential relationship did exist with some of the stronger athletes at baseline demonstrating greater performance effects whilst the weaker athletes at baseline experienced negative effects in response to a cycle sprint pre-conditioning exercise. The efficacy of the two exercise strategies appeared to be governed to therefore by the neuromuscular thresholds (e.g. strength status) specific to each athlete as well as protocol design characteristics (e.g. exercise selection, set and repetition volume and recovery duration). Consequently, the training design considerations that promote PAP and avoid prolonged periods of fatigue may be highly specific to each individual athlete. It is also apparent that manipulating free-testosterone concentrations via an exercise pre-conditioning routine did not support functional improvements in whole body force and power expression during habitual training practice in elite rugby union players. A cycle sprint pre-conditioning strategy for the purpose of elevating

hormones could induce fatigue and limit muscle potentiation regardless of any acute increases in circulating free-testosterone.

5.6 Practical recommendations

For a pre-conditioning exercise to be effective, it is recommended that strength and conditioning coaches first establish the conditions that support dynamic performance on an individual athlete basis. This may include assessment of the pre-conditioning exercise mode, intensity, volume and recovery duration time frames which provides muscle excitation but avoid fatigue. Establishing these characteristics for each athlete in a time-limited team sport environment may not however be practical during applied training periods. Designing pre-conditioning exercises to initiate hormonal elevations could negatively influence training output and therefore priming hormones through physical warm-up routines must be approached with caution. It is recommended that strategies which elevate hormones through non-physical means (i.e. psychological stimulation) be investigated further if the non-genomic effects of hormones are to be realised in an applied training environment.

CHAPTER 6: THE EFFECT OF WORK INTERVAL DURATION ON POWER AND MUSCLE FIBRE ACTIVATION DURING HIGH-INTENSITY INTERVAL TRAINIING (HIT)

6.1 Introduction

Intermittent team sports require repeated execution of high power efforts interspersed with brief recovery intervals over an extended period of time (Cahill et al., 2013, Roberts et al., 2008). Consequently, identifying exercise strategies that improve repeated force production under conditions of fatigue is an important training consideration for elite team sport athletes and conditioners. Studies within this thesis have examined chronic (Chapter 3 and Chapter 4) and acute (Chapter 5) methods for increasing the expression of maximal strength and power. The final experimental study was therefore focused on training designs which improve an athlete's muscle endurance potential.

High-intensity interval training (HIT), which is defined as repeated work intervals of maximal or near maximal exercise interspersed with short recovery periods (Gibala, 2009), is a commonly used exercise format in professional training environments. However, specific recommendations regarding the HIT programme design characteristics that influence short-term high intensity intermittent exercise capacity and muscle recruitment in elite athletes is currently lacking.

Optimising the intensity of HIT strategies through the manipulation of work to relief interval duration is an important programme design consideration as physiological and performance adaptations depend on the accumulated load completed at high exercise intensity (Castagna et al., 2011, Buchheit and Laursen, 2013b). Consequently, whilst several HIT formats exist, repeated sets of short work intervals (i.e. < 60 seconds, 'short HIT') have been reported to accumulate a greater quantity of total work at higher exercise intensities, and to provide greater training specificity for intermittent team sports, when compared with longer HIT work intervals (i.e. > 60 seconds) (Girard et al., 2011, Buchheit and Laursen, 2013b, Ronnestad et al., 2014). Short HIT has been associated with improved endurance performance by initiating positive adaptations in skeletal muscle oxidative capacity (Laursen and Jenkins, 2002, Burgomaster et al., 2005, Buchheit and Laursen, 2013b, Jacobs et al., 2013). In particular, the high intensities achieved via short HIT have been reported to promote superior activation of signalling pathways that control mitochondrial biogenesis (Edgett et al., 2013, Ronnestad et

al., 2014). Short HIT also appears to facilitate preferential recruitment of larger motor units due to elevated neuromuscular engagement of type II muscle fibre (Altenburg et al., 2007, Buchheit and Laursen, 2013b). Increasing the recruitment capacity of type II muscle fibre through short HIT may therefore be an important factor driving endurance adaptation potential in muscle fibres that are deemed to be of value to athletes who require repeated power efforts.

The proposed effectiveness of short HIT to initiate high muscle activation may be associated with the force and velocity demands of the exercise. Short work bouts are characterised by frequent accelerations, decelerations and re-accelerations and are generally performed at greater absolute muscle contraction speeds (Buchheit and Laursen, 2013a). As short HIT requires high muscle forces during fast contraction velocities, a greater number of motor units that are more accomplished at producing high power outputs (e.g. type II motor units) may become engaged (Croce et al., 2014). Since a fibre must be recruited to initiate an adaptive response (Schoenfeld et al., 2014), it seems logical to suggest that shorter work bouts may initiate higher accumulative neuromuscular load in fast muscle fibre when compared with longer bout HIT strategies (Buchheit and Laursen, 2013a).

Coaches in applied sport settings regularly use short HIT protocols, but there is little data available detailing the effect of work interval durations lasting less than 30 seconds (using a fixed work/relief ratio) on muscle fibre specific activity. Subtle manipulations in these programme design characteristics are often used under the assumption the manipulations will initiate progressive response pathways in elite team sport athletes; however, it is not known, for example, whether a HIT session incorporating repeated 10 second work periods enables greater accumulated training intensity and neuromuscular engagement than a session with 20 second work periods.

The aim of this study was to analyse the acute effects of three HIT protocols, differentiated by work to relief interval duration while maintaining a fixed exercise volume, on power and muscle fibre specific activity in professional rugby union players. It was hypothesised that the shortest HIT work to relief protocol would initiate the highest training power outputs, which would be associated with a greater accumulation of time activating higher threshold motor units.

6.2 Materials and methods

6.2.1 Experimental Design

This study was a repeated measures design to identify the effect of HIT work to relief interval configuration on power and muscle fibre recruitment patterns during cycle ergometer training.

The experimental design consisted of three HIT sessions performed by each participant, with each session separated by a week. Each protocol was standardised for total work and relief interval duration (240 s and 480 s respectively), but differed in distribution of work and relief interval time frames. The three cycle HIT protocols comprised: a) 24 efforts of 10 s work to 20 s relief intervals ('10-20'), b) 16 efforts of 15 s work to 30 s relief intervals ('15-30'), and c) 12 efforts of 20 s work to 40 s relief intervals ('20-40'). A random number generator (Microsoft Office Excel) was used to allocate each participant to one of the three protocols for the first testing session. The same process was repeated for the second session but with the previously allocated protocols removed, whilst the third testing session was prescribed based on the protocol which remained for each participant. Each HIT session was performed on the same day of the week between 08:00 and 11:00 hours. Short HIT training formed a regular component of participants' weekly training programme, therefore each athlete was familiar with this exercise.

6.2.2 Participants

Ten first team professional rugby union athletes from an English premiership rugby team volunteered to take part in this study. Each athlete had at least two years of short HIT experience, was over the age of 18 and provided written informed consent. Two participants were removed from analysis due to failure to complete all three experimental sessions; subsequently eight participants were analysed in the current study (age, 23 ± 1; height, 1.89 ± 0.10 m; body mass, 107.8 ± 10.0 kg). Study procedures were approved by an institutional ethics committee (Research Ethics Advisory Committee for Health, University of Bath).

6.2.3 Cycle HIT protocols and power measurement

The short HIT protocols were informed by evidence that repeated efforts of short work bouts (10 to 20 s) with short relief periods (20 to 40 s), deliver an accumulated training intensity sufficient to elicit high anaerobic glycolytic and neuromuscular strain (Buchheit and Laursen, 2013b). The HIT protocols were performed on a Wattbike (Wattbike Pro, Wattbike Ltd, Nottingham, UK). Seat height was adjusted and standardised for each individual and participants remained seated throughout all cycle exertions. Each participant performed all cycling bouts against the same absolute air (level 8) and magnetic resistance (resistance 1). Measures of power were recorded via the Wattbike load cell measurement system at a resolution of 100 samples per second.

Each HIT protocol consisted of a brief warm up (five minute continuous cycle at 40 W), followed by a 6-second baseline maximum cycle sprint which served as the reference point for power and muscle fibre activation during efforts for each HIT protocol. During baseline

maximum tests, participants were instructed to cycle as hard as possible (i.e. 'all-out') for six seconds. Mean and peak power was recorded after the completion of each baseline maximum. A four-minute rest period was given after each baseline maximum to minimise the effect of fatigue on the subsequent HIT repetitions (Croce et al., 2014). The HIT repetitions were performed maximally, and participants were instructed to produce as much power as possible throughout the duration of each repetition. Each participant was aware of the total number of efforts associated with each HIT protocol. The Wattbike performance computer provided the participants with feedback throughout each HIT work effort. After each HIT effort, average and peak power were recorded. Strong verbal encouragement was give throughout all baseline maximum and HIT cycle protocols. Between session reliability was assessed for the three baseline maximum measures of average and peak power. For measures of average power the ICC was 0.75 and the CV was 5.4%, for measures of peak power the ICC was 0.62 and the CV was 6.9%.

6.2.4 EMG measurement and analysis

To measure the myoelectrical activity of each cycle baseline maximum and HIT effort, wavelet transform analysis of surface electromyography (SEMG) data from the vastus lateralis (VL), rectus femoris (RF) and lateral gastrocnemius (LG) of the right leg was performed. Wireless SEMG electrodes were attached to the RF, VL and LG muscles of the right leg. Before sensor placement, hair was shaven and the skin cleaned with an alcohol swab to minimise impedance. The wireless SEMG electrodes (Trigno, Delsys Inc; Boston, MA, USA) were positioned in the centre of the muscle belly parallel to the presumed orientation of the muscle fibres according to SENIAM guidelines (SENIAM, 2005) and applied using specialised double-sided adhesive (Trigno adhesive, Delsys). To control for reproducibility error of electrode placement, all SEMG signals collected during each separate HIT protocol was compared against the same testing session's baseline maximum values. Consequently, the same orientation of electrode placement was used during baseline testing and HIT protocol assessments. SEMG signals were pre-amplified (×100), amplified (×2), band-pass filtered (10-1000 Hz), and sampled at 2500 Hz with EMG Works software (version 4.0, Delsys, Boston, MA, USA).

In order to identify activation patterns of fast and slow motor units, the SEMG signals from the RF, VL and LG muscles were resolved into their myoelectrical intensities as a function of time and frequency using wavelet transform techniques (von Tscharner, 2000). Each myoelectrical signal was decomposed in the time-frequency domain using a set of 11 nonlinearly scaled wavelets. Each wavelet domain was characterised by its centre frequency and time resolution (So et al., 2009, von Tscharner, 2000). Centre frequency is defined as the position of the

maximum of the wavelet in the frequency space (von Tscharner, 2000). Details of centre frequency and associated time resolution for the 11 wavelet domains are listed in Table 6.1. Each wavelet was convolved with the SEMG. The convolved signals were converted back to time domain by the inverse Fast Fourier transform (So et al., 2009). The power intensity pattern for each wavelet domain within each baseline maximum and HIT effort cycle bout was subsequently obtained. The intensity represented the timing and strength of the muscle activation from different types of motor units at various frequencies (Hodson-Tole and Wakeling, 2009, So et al., 2009). The first and last wavelet domains (wavelets 0 and 10) were excluded from further analysis due to low and high frequency noise (Hodson-Tole and Wakeling, 2008). The frequency band 19-62 Hz (wavelets 1-3) was characterised as signals from slow motor units; the frequency band 218-331 Hz (wavelets 7-9) was characterised as signals from fast motor units, whilst total frequency was identified between frequency band 19-331 Hz (wavelets 1-9). The mean signal intensity for wavelet domain 7-9 was calculated for each baseline maximum and HIT cycle efforts. Wavelet energy in this domain was defined as fast muscle activation and subsequently used for all comparative analysis between HIT protocols for each muscle. Data analysis was performed using MATLAB with the wavelet toolbox (Version 7.1, The MathWorks, Natick, MA, USA).

Table 6.1 Centre frequency and time resolution for the 11 wavelet domains.

Wavelet number	0	1	2	3	4	5	6	7	8	9	10
Centre frequency (Hz)	7	19	38	62	92	128	170	218	271	331	395
Time resolution (ms)	77	59	41	32	26	22	20	17	15	14	12

6.2.5 Data processing

Mean power and peak power from the Wattbike, and fast muscle activation for the RF, VL and LG were recorded as a mean percentage value relative to the baseline maximum trial over each HIT protocol efforts. To allow for comparison between the three HIT protocols, the mean percentage values relative to the baseline maximum for each measure was identified at five common time points (Table 6.2). The mean value of each measure over the five time points was then used to represent the total performance measure over the 240 s exercise period for each HIT protocol.

Table 6.2 The five efforts and corresponding time points selected for comparison between each HIT protocol.

	10-20		15-30		20-40	
Time point	Effort number	Work duration (s)	Effort number	Work duration (s)	Effort number	Work duration (s)
1	1	0-10	1	0-15	1	0-20
2	6	60-70	4	60-75	3	60-80
3	12	120-130	8	120-135	6	120-140
4	18	180-190	12	180-195	9	180-200
5	24	230-240	16	225-240	12	220-240

6.2.6 Statistical analysis

Statistical analysis was conducted using IBM SPSS Statistics (version 22, SPSS Inc, Chicago, IL, USA). All values were represented as Mean ± SD and statistical significance was taken at $p \leq 0.05$. A two-way ANOVA with repeated measures was used to identify if significant main effects existed over the five selected time points and between HIT protocols for the dependent measures of fast muscle activation, peak power and mean power. The interaction effects (time x protocol) were also considered. All the data were tested for sphericity and paired t-tests were used for post hoc analysis. To identify the magnitude of difference in fast muscle activation, peak power and mean power between the three HIT protocols, effect sizes were calculated between the average values over the five time points. Differences in fast muscle activation, peak power and mean power were categorised as trivial (ES <0.2), small (ES 0.2-0.6), moderate (ES 0.6-1.2), large (ES 1.2-2.0) and very large (ES >2.0) (Hopkins, 2004). In order to identify the uncertainty around the true value of the reported effect size statistics, 90% confidence intervals around each effect size were reported based on the methods of Hopkins (2007). Subsequently, magnitude based clinical inferences were attached to each effect size and reported as the percentage likelihood, with descriptor, that the true effect was positive, uncertain or negative in favour of a given HIT protocol (Hopkins, 2007). For an effect size difference between protocols to be clear, the odds of benefit relative to odds of harm (odds ratio) had to be > 66 (Hopkins, 2007).

6.3 Results

6.3.1 Power

There were no significant time × protocol interaction effects for measures of mean or peak power (Table 6.3 A, B). A significant protocol effect was observed for mean power ($p = 0.01$, Table 6.3 A, B). Post hoc analysis identified a higher mean power in the 10-20 versus the 20-40 protocol with this difference deemed very likely beneficial in favour of the 10-20 protocol (+11%, $p = 0.02$, ES = 0.55 ± 0.3, Figure 6.1 A). Post hoc analysis also identified a higher mean power in the 15-30 versus the 20-40 protocol with this differences also deemed very likely beneficial in favour of the 15-30 protocol (+5%, $p = 0.01$, ES = 0.28 ± 0.1, Figure 6.1 C). A significant time effect was identified in mean and peak power. Post hoc analysis revealed decrements in mean ($p = 0.01$) and peak ($p = 0.01$) power over the five time periods irrespective of HIT protocol. Possible beneficial differences in mean power in favour of the 10-20 protocol was observed against the 15-30 protocol (+6%, $p = 0.10$ [paired t-test], ES = 0.31 ± 0.3, Figure 6.1 B). Possible beneficial differences in peak power in favour of the 10-20 protocol was also observed against the 20-40 protocol (+7%, $p = 0.03$ [paired t-test], ES = 0.35 ± 0.2, Figure 6.1 A) and the 15-30 protocol (+6%, $p = 0.03$ [paired t-test], ES = 0.35 ± 0.2, Figure 6.1 B), respectively.

6.3.2 Muscle activation

There were no significant time × protocol interaction effects or significant main effects for protocol for fast muscle activation (Table 6.4 A, B, C). A significant time effect was identified in fast muscle activation. Post hoc analysis revealed decrements in VL ($p = 0.01$) and LG ($p = 0.04$) fast muscle activation over the five time periods irrespective of HIT protocol.

Very likely beneficial differences in fast activation of the RF in favour of the 10-20 protocol was observed against the 20-40 protocol (+24%, $p = 0.02$ [paired t-test], ES = 1.40 ± 0.9, Figure 6.1 A), whilst very likely beneficial differences in favour of the 15-30 protocol was also observed against the 20-40 protocol (+19%, $p = 0.05$ [paired t-test], ES = 1.05 ± 0.8, Figure 6.1 C). A possibly beneficial difference in fast activation of the RF in favour of the 10-20 protocol was observed against the 15-30 protocol (+6%, $p = 0.04$ [paired t-test], ES = 0.26 ± 0.2, Figure 6.1 B).

Between protocol differences in fast muscle activation of the VL were small, with a possible negative difference in the 10-20 protocol against the 20-40 protocol (-8%, $p = 0.09$ [paired t-test], ES = -0.23 ± 0.2, Figure 6.1 A), and 15-30 protocol (-9%, $p = 0.03$ [paired t-test], ES = -

0.23 ± 0.2, Figure 6.1 B), respectively. The difference between the 15-30 and 20-40 protocol was unclear (+1%, p = 0.81 [paired t-test], ES = 0.04 ± 0.3, Figure 6.1 C).

A likely negative difference in LG fast activation was observed in the 10-20 against the 20-40 protocol (-11%, p = 0.02 [paired t-test], ES= -0.53 ± 0.3, Figure 6.1 A). A most likely negative difference in LG fast activation was identified in the 10-20 against the 15-30 protocol (-50%, p = 0.01 [paired t-test], ES = -2.10 ± 0.6, Figure 6.1 B). A very likely beneficial difference in LG fast activation in favour of the 15-30 protocol was observed against the 20-40 protocol (+26%, p = 0.01 [paired t-test], ES = 1.62 ± 0.5, Figure 6.1 C).

Table 6.3 Power over the five common time points and as a mean of the five time points between the three HIT protocols. (A) Mean power, (B) peak power. Values are represented as a percentage relative to the baseline maximum reference value (Mean ± SD).

A)

Time Point[##]	10-20 (%)	15-30 (%)	20-40 (%)
1	69.9 ± (6.7)	60.8 ± (8.2)	58.5 ± (8.5)
2	51.0 ± (3.5)	48.8 ± (5.7)	47.1 ± (4.8)
3	44.8 ± (3.8)	43.6 ± (6.0)	41.5 ± (4.6)
4	42.4 ± (4.3)	41.4 ± (5.7)	39.3 ± (4.4)
5	44.5 ± (6.0)	42.1 ± (8.9)	38.8 ± (5.5)
Mean	50.5 ± (11.3)[**]	47.3 ± (8.0)[*]	45.0 ± (8.2)

[##]Significant time effect over the 5 time points $p < 0.01$,

**Significantly different to 20-40 protocol mean power $p < 0.01$,

*Significantly different to 20-40 protocol mean power $p < 0.05$

B)

Time Point[##]	10-20 (%)	15-30 (%)	20-40 (%)
1	82.7 ± (9.5)	74.3 ± (9.4)	73.6 ± (6.3)
2	64.1 ± (8.0)	61.5 ± (6.1)	61.0 ± (11.8)
3	54.5 ± (6.5)	52.4 ± (4.6)	53.5 ± (9.9)
4	53.5 ± (10.2)	49.0 ± (8.4)	50.2 ± (12.5)
5	53.6 ± (11.0)	51.6 ± (12.3)	48.1 ± (8.5)
Mean	61.7 ± (12.6)	57.7 ± (10.4)	57.3 ± (10.4)

[##]Significant time effect over the 5 time points $p < 0.01$

Table 6.4 Fast muscle activation over the five common time points and as a mean of the five time points between the three HIT protocols. (A) Rectus femoris, (B) vastus lateralis, (C) lateral gastrocnemius. Values are represented as a percentage relative to the baseline maximum reference value (Mean ± SD). Fast muscle activation = Mean signal intensity of wavelets recorded between frequency bands 218-331 Hz (wavelets 7-9).

A)

Time Point	10-20 (%)	15-30 (%)	20-40 (%)
1	45.0 ± (24.3)	44.1 ± (27.0)	30.5 ± (11.5)
2	36.4 ± (22.0)	32.1 ± (18.6)	25.9 ± (17.7)
3	30.1 ± (17.8)	29.1 ± (21.9)	24.4 ± (17.6)
4	24.5 ± (13.2)	23.3 ± (14.4)	23.2 ± (14.3)
5	34.3 ± (20.2)	32.0 ± (21.4)	26.3 ±(17.4)
Mean	34.1 ± (7.6)	32. 1 ± (7.6)	26.0 ± (2.8)

B)

Time Point[##]	10-20 (%)	15-30 (%)	20-40 (%)
1	57.9 ± (31.5)	63.7 ± (6.3)	55.8 ± (21.4)
2	34.4 ± (21.1)	34.6 ± (4.6)	37.6 ± (16.8)
3	29.5 ± (22.8)	31.0 ± (10.5)	33.5 ± (9.6)
4	27.8 ± (25.3)	31.3 ± (12.3)	31.5 ± (7.9)
5	28.7 ± (30.4)	33.2 ± (14.4)	33.0 ± (8.9)
Mean	35.6 ± (12.7)	38.7 ± (14.0)	38.3 ± (10.1)

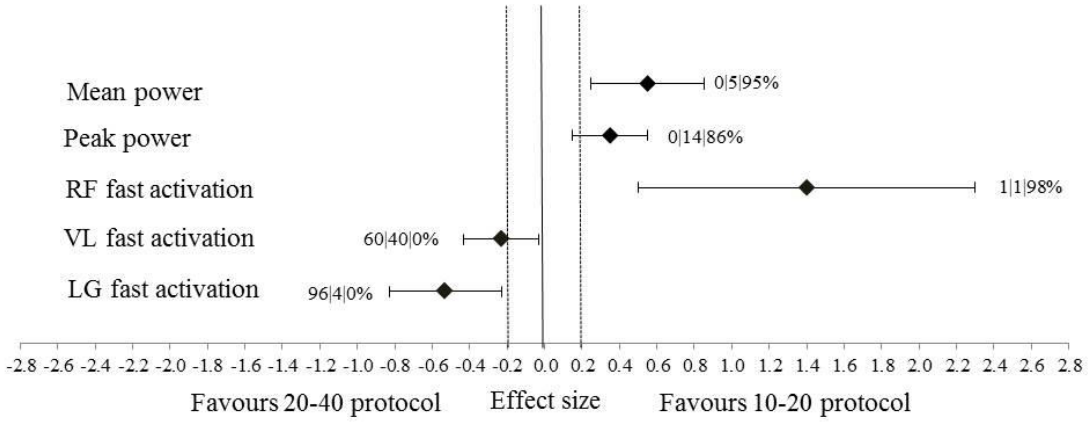
[##]Significant time effect over the 5 time points $p < 0.01$

C)

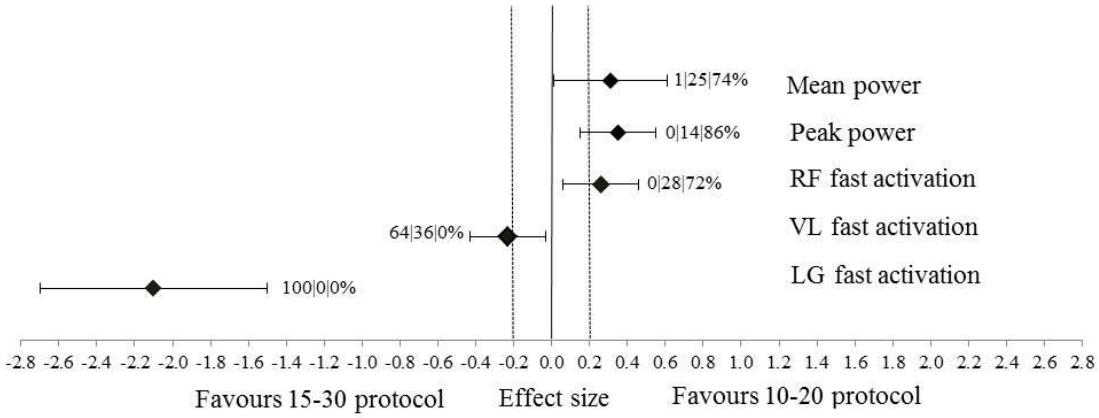
Time Point[##]	10-20 (%)	15-30 (%)	20-40 (%)
1	55.0 ± (16.9)	80.7 ± (53.1)	59.2 ± (31.4)
2	43.8 ± (15.8)	58.1 ± (37.8)	49.2 ± (37.8)
3	34.7 ± (10.5)	56.1 ± (25.0)	43.7 ± (28.8)
4	34.3 ± (15.5)	55.7 ± (17.3)	37.0 ± (16.5)
5	38.1 ± (11.8)	57.4 ± (25.7)	40.1 ± (18.1)
Mean	41.1 ± (8.6)	61.5 ± (10.7)	45.8 ± (8.7)

[##]Significant time effect over the 5 time points $p < 0.05$

A)



B)



C)

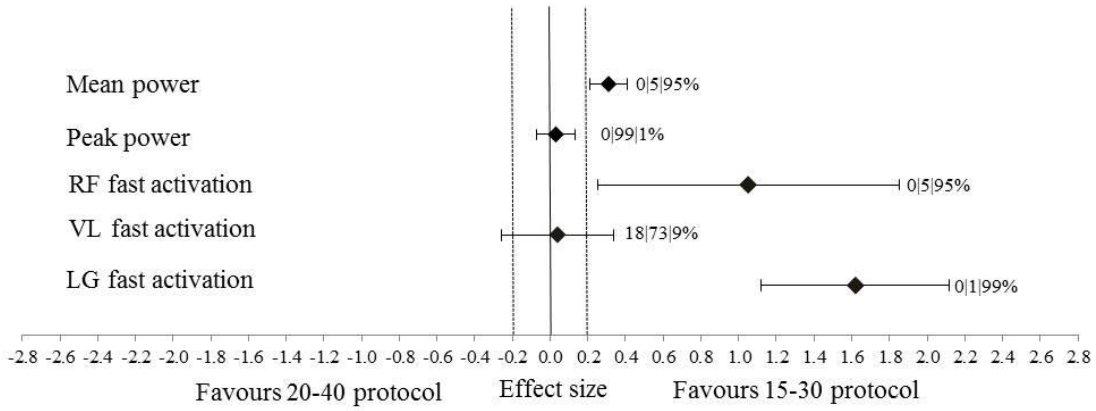


Figure 6.1 Forest plot representing the magnitude of effect size difference (± CI) in power and fast muscle activation between the 10-20 and 20-40 protocols (A), 15-30 and 10-20 protocols and (C) 20-40 and 15-30 protocols. Data labels give likelihoods that the effect size difference is substantially negative | uncertain | positive in favour of the 10-20 protocol (A and B) and in favour of the 15-30 protocol (C). RF = rectus femoris, VL = vastus lateralis, LG = lateral gastrocnemius.

6.4 Discussion

This study evaluated the acute effects of manipulating HIT work interval duration on power measures and fast muscle activation in elite rugby union players. Large beneficial increases in mean and peak power were identified in response to the 10-20 and 15-30 protocols relative to the 20-40 protocol. Fast muscle activation in the RF was also elevated in response to both the 10-20 and 15-30 protocol, whilst LG fast activation was greatest in response to the 15-30 protocol. These findings suggest shorter work interval durations (e.g. 10 or 15 s) appear to support greater total power and high threshold motor unit engagement in selected muscles during HIT protocols matched for total exercise volume.

The significant effects for time irrespective of the design differences between HIT protocols demonstrates that all three strategies created conditions where progressive decrements in power and muscle activation occurred. However, it was the shorter HIT protocols which maintained measures of mean and peak power. The superior power in response to the shorter HIT protocols supports previous research analysing the neuromuscular response patterns to HIT (Edgett et al., 2013, Ronnestad et al., 2014). Edgett et al. (2013) demonstrated significantly greater mean power, muscle activation and mitochondrial biogenesis gene

expression when work matched cycle HIT was performed with maximal exercise intensities (i.e. 100% peak aerobic power) in comparison to submaximal (i.e. 77% peak aerobic power). This suggests the magnitude of muscle power and intracellular adaptive response pathways to an acute HIT protocol may be intensity dependent. The present findings also demonstrate that a single session of HIT designed with multiple efforts of shorter work intervals facilitates significantly greater total amounts of high intensity work (i.e. mean power increased between 5-11%) than longer work intervals performed with fewer efforts.

Large beneficial increases in fast muscle activation in the RF in response to the 10-20 and 15-30 protocols were also recorded when compared with the 20-40, with the largest effect identified in the 10-20 protocol. The elevation in power and greater RF fast muscle recruitment suggests the 10-20 protocol elicited different mechanical demands to the 20-40. Motor unit recruitment patterns during cycling at a fixed load but with a range of pedalling speeds have demonstrated a preferential recruitment effect occurs for faster motor units as pedalling velocity increases (Wakeling et al., 2006). This has been associated with the higher firing rates and conduction velocities which characterise the intrinsic properties of fast motor units (Hodson-Tole and Wakeling, 2009). It may be speculated that the contractile requirements of the shorter intervals created a mechanical demand which selectively increased the recruitment of faster motor units in the RF. This demand may be associated with the greater number of total acceleration phases performed in the shorter protocols (due to the greater total number of efforts [Table 6.2]) which could increase the requirement for muscle force production under high contraction velocities (Buchheit and Laursen, 2013a).

This is the first study to report differences in muscle activation as a function of exercise intensity during a training protocol designed to improve localised muscular endurance. Strength training studies have reported significant elevations in muscle activation of the quadriceps femoris in response to higher intensity load prescriptions (Akima and Saito, 2013). In particular, Schoenfeld et al. (2014) found that muscle contractions performed at 70% 1 repetition maximum (1RM) provided greater total average (+35%) and peak (+22%) quadriceps muscle activation when compared to 30% 1RM, with the observed differences attributed to the greater force demands of the 70% protocol. In a similar manner, muscle fibre recruitment patterns in response to a HIT session also appear to be influenced by the intensity requirements of the exercise. Consequently, shortening work interval durations appears to be an effective programme design strategy for elevating the intensity and recruitment potential of fast muscle fibres during a HIT session.

Fast muscle activation in the VL demonstrated the lowest between intervention response patterns, with no likely differences occurring between the three HIT protocols. Whilst clear protocol differences existed in RF fast muscle activation, the VL data reflects the variation that exists in recruitment responses between quadriceps muscles. Croce et al. (2014), for example, demonstrated significantly elevated fast muscle activation in the RF relative to the VL at progressively greater knee extension contraction intensities (25-100% of maximum voluntary contraction). These discrepancies may be explained by the higher percentage of type II muscle fibre in the RF compared with the VL (Johnson et al., 1973, Polgar et al., 1973) and a progressive recruitment of these fibres in response to the greater intensity demands of the shorter HIT protocols, since SEMG frequencies are influenced by muscles with a greater percentage distribution and diameter of type II muscle fibre (Croce et al., 2014, Gerdle et al., 2000).

The LG fast activation demonstrated extreme response patterns, with large beneficial effects in favour of the 15-30 protocol when compared to the 10-20 and 20-40. The fact that the LG did not follow a similar trend in activation to the RF was unexpected. During the baseline maximum conditions, SEMG frequencies of the LG demonstrated lower relative contributions (<10%) to total muscle activation (i.e. LG, RF and VL combined). Consequently, the volatile response patterns of the LG may reflect the lower activation capacity of this muscle during the three HIT protocols. Wakeling et al. (2006) also recorded lower relative EMG frequencies in LG during cycling at progressive intensities (i.e. increased pedal cadence), but reported greater fast fibre recruitment due to greater fascicle strain rates in the medial gastrocnemius. It may be speculated that the mechanical requirement of the exercise modality in the current study provided sub-optimal contractile conditions for consistent recruitment patterns in the LG to be realised. Future studies may look to select more than one muscle of the triceps surae to give a clearer picture of fast activation potential of this muscle group during cycle HIT exercise.

Finally, it may be speculated that the lower power responses observed in the 20-40 protocol is related to the adoption of pacing. Lower peak and mean power outputs have been reported during the first 10 s of a 30 and 45 s all-out cycle test when compared with a 15 s all-out cycle test (Wittekind et al., 2011). Consequently, pacing may occur during the initial stages of maximal cycling when work periods exceed 15 s in duration (Wittekind et al., 2011). HIT strategies employing work periods greater than 15 s could therefore be more susceptible to the centrally controlled regulatory effects of pacing. As the participants in the present study had

full knowledge of the HIT work durations, the dampened power responses from time point 1-5 (Table 6.3) in the 20-40 protocol may reflect this theory.

6.4.1 Limitations

Whilst the present study identifies intensity dependent response patterns in power and fast muscle activation in selected muscles, it is unknown whether these acute responses will translate into greater intrinsic adaptation in type II muscle fibre. Further work is required to determine the extent to which HIT programmes designed with shorter intervals support elevated chronic training adaptation. It must also be recognised that different motor tasks will influence the pattern and magnitude of motor unit activity within the same group of muscles (Hodson-Tole et al., 2013). Consequently, extrapolating the fast muscle activation patterns identified in the present study to other forms of HIT performed with different exercise modes should be approached with caution. Analysis into muscle activation patterns during HIT performed when running with changes of direction may be a pertinent area of future research for team sport athletes.

6.5 Conclusions

In the present study, short work intervals increased net fast motor unit activation in selected muscle groups and provided a greater potential to repeatedly generate higher power in elite rugby players. In particular, fast motor units of the RF appeared to be recruited in a task specific fashion in response to the elevated mechanical demands of the shorter HIT protocols. Manipulating HIT work interval duration may therefore have implications for the chronic development of fatigue resistance in muscle fibres (e.g. type II) which have a greater capacity to generate high power contractions. These findings are important for athletes whose performance outcomes are related to their ability to repeatedly perform high-intensity intermittent exercise.

6.6 Practical applications

Athletes looking to maximise muscular engagement and performance responses to an acute cycle HIT session may benefit from reduced work interval duration of the exercise protocol (e.g. 10 or 15 s) whilst maintaining total exercise volume. A work to relief ratio of 1:2 and a fixed total work volume of 240 s provides an initial framework from which HIT can be implemented and progressed in an applied environment. Manipulating work interval duration

within this framework will provide coaches in elite team sport settings with an effective method for increasing the acute and chronic intensity prescription and fibre specific adaptation potential of a training session.

CHAPTER 7: GENERAL DISCUSSION

7.1 Addressing the research objectives

This thesis was conducted to improve the understanding of strength and power training strategies which promote physical development in elite rugby union athletes. To achieve this overall aim a number of specific objectives were established and addressed briefly in this section prior to a broader interpretation of the research in section 7.2.

Objective 1) Monitor strength and power development together with retention and decay potential over pre-season, mid-season and end of season training phases in the English premiership;

Key Findings (Chapter 3):

- Beneficial increases in peak force, early force and power was realised over pre and mid-season training phases.
- Maintenance in peak force and power and small declines in early force production was demonstrated toward the end of season phase.
- The net effect of a full season on measures of peak force, early force and power appears positive.

Objective 2) Investigate the effects of implementing a complex training programme on measures of strength, power and speed over an in-season training cycle;

Key Findings (Chapter 4):

- Complex training was effective at developing selective measures of power whilst maintaining strength during an in-season training phase.
- Complex and strength training alone was not sufficient to promote improvements in speed.

Objective 3) Examine salivary steroid hormonal response patterns to complex and strength training strategies over an in-season training cycle;

Key Findings (Chapter 4):

- The complex and strength training interventions demonstrated no clear effects on acute (i.e. pre to post exercise) and chronic (i.e. baseline resting) alterations in testosterone and cortisol.
- No associations were realised between training mediated changes in performance (i.e. strength and power) and hormones.

Objective 4) Identify the acute potentiating effects of two physical pre-conditioning strategies on subsequent measures of power;

Key Findings (Chapter 5):

- No group improvements in power were realised in response to a weightlifting (leg press) or cycle sprint pre-conditioning strategy.
- Individual variation in dynamic performance in response to pre-conditioning exercise may be related to the base strength of an athlete.

Objective 5) Investigate associations between exercise induced alterations in blood concentrations of steroid hormones and power;

Key Findings (Chapter 5):

- The cycle sprint protocol significantly elevated free-testosterone concentrations.
- Elevated free-testosterone was not associated with local or systemic muscle power.

Objective 6) Identify the effect of work interval duration manipulation on fast muscle fibre activity and power during short HIT exercise.

Key Findings (Chapter 6):

- HIT protocols configured with shorter work interval durations initiated greater total power responses and fast muscle fibre activation.

7.2 Contribution to knowledge

There is a lack of robust scientific evidence detailing the capacity for lower body strength and power development in rugby union players competing in the English premiership over pre and in-season training phases. The findings from Chapter 3 demonstrate the greater opportunity pre, early and mid-season training phases represent for the development of strength and power. This study also highlights the limited potential for physical enhancement during the final stages of a professional season. It was initially hypothesised that drop off in physical

performance during this period would not occur. However, this was based on the assumption that external training loads (i.e. rugby training volume and intensity) would decrease or "taper" towards the end of the season. This did not occur with the current rugby team. As the end of season phase for professional rugby teams is the most important period of the competitive year (e.g. play off and grand final matches), it seems appropriate that coaches put in place strategies which maximise the potential for physical maintenance. These may include alterations to weekly scheduling to provide longer recovery intervals between match play and training, reduced rugby training frequency, or physical development weeks where players are rested from match play and strength and power training is emphasised over technical and tactical training.

Chapter 3 is the first investigation to demonstrate positive net increases in measures of strength and power over a full competitive season in elite rugby players competing in the English premiership. The physical development reported over this period illustrates the substantial time frames required for training adaptation to be realised in this athletic population. This finding reflects the diminished scope for strength and power improvements indicative of elite athletes as well as the negative effects concurrent training and match play loads can have on physical performance. Subsequently, physical tracking in this environment should be conducted over long-term periods, with measures of strength and power tested numerous times over multiple performance cycles to account for the different physical outcomes associated with each phase of a season.

Having established that maximum strength and power can be improved in a professional rugby environment but that these improvements are difficult to achieve, complex training was investigated in both chronic (Chapter 4) and acute (Chapter 5) contexts. A novel approach for both studies was the inclusion of hormone monitoring; it was expected that hormone response patterns would be associated with any observed performance responses to both the complex training study and the acute response study.

Since the mid-season phase in professional rugby union represents a time when meaningful improvements in power can be gained (Chapter 3), Chapter 4 analysed the efficacy of a chronic complex training intervention on strength, power and speed during this period. This training study demonstrates complex training has the potential to support improvements in power to a greater degree than strength training alone in young elite rugby players when implemented into an in-season training programme. As a clear relationship between strength and power exists (Cormie et al., 2010b) and was identified in Chapter 3 at mid-season testing,

complex training could serve as a useful short-term training tool (i.e. 4-5 week concentrated blocks) to provide a means for increasing power whilst stabilising strength. Chapter 4 also provides evidence to suggest weekly training programmes should include training phases when the primary focus of adaptation is on maximum strength development. The protocol cycled complex training followed by strength training blocks and demonstrated significant improvements in all measures of strength and power when this sequence was periodised over the 10-week intervention period. It is therefore advisable that training cycles switch between complex "power" focused phases followed by strength focused phases in order to initiate a wide spectrum of neuromuscular adaption potential.

Chapter 4 demonstrated the importance of training specificity for elite athlete groups as measures of speed (which was not a training component of either intervention) reported a trend towards inferior performance. Increasing maximum force via strength training appears to initiate a global improvement in physical performance across a wide spectrum of the force-velocity continuum (e.g. power and speed) in untrained athletes (Cormie et al., 2010b, Cormie et al., 2010a). However, Chapter 4 results demonstrate that increasing the neuromuscular factors that underpin strength and power through a combination of complex and traditional strength training will not translate into functional gains in speed in elite rugby players. The mechanical demands of sprinting should therefore be included in a complex set or alongside resistance training sessions for the enhancement of this capacity to be realised. The athletes in Chapter 4 were senior academy players with a slightly lower training age in comparison to the players selected in Chapters 3, 5 and 6. It was therefore interesting to discover that even young strength trained athletes require a varied and specific training approach if strength, power and speed are to be improved.

When monitored over short (4 weeks) and longer training periods (10 weeks) the hormonal response patterns in Chapter 4 were unclear. With the equivocal findings of a long-term role for hormones in support of training adaptation, Chapter 5 looked to determine if a short-term "non-genomic" role for hormones existed. The elevated free-testosterone in response to the cycle sprint protocol supports other evidence demonstrating metabolically taxing exercise routines which generate high lactate levels are more effective at initiating steroid response patterns (Smilios et al., 2003, Obminski et al., 1998). This offers further reasoning as to why unclear testosterone response patterns were reported in Chapter 4, as both complex and strength training protocols were performed at high exercise intensity but with low training volume. However, the increased availability of biologically available testosterone reported in Chapter 5 did not facilitate acute improvements in lower or upper body dynamic performance.

The lack of control of external exercise stressors in Chapter 4 and Chapter 5 could have influenced the acute and chronic testosterone and cortisol responses. Consequently, the current elite rugby setting provided an unpredictable environment for monitoring testosterone and cortisol. This is not to say that chronic and acute manipulation of hormones do not support training adaptation or short-term neuromuscular potentiation in sporting environments where, i) athletes present more homogenous physical characteristics, and ii) the weekly load of match play and technical training is more repeatable. However, strength and conditioning coaches in applied rugby union environments cannot control the weekly training and match play factors which precede strength and power workouts. As training adaptations were realised in Chapter 4 irrespective of a clear hormonal influence, and free-testosterone did not appear to support lower or upper body power in Chapter 5, then the role of monitoring hormones in this professional setting appears difficult to clarify.

Chapter 5 also highlighted the caution strength and conditioning coaches must exercise when selecting and programming pre-conditioning routines. The cycle sprint protocol, whilst effective at elevating free-testosterone, had a detrimental effect on subsequent dynamic performance within the weaker athletes in the group, indicating fatigue and not muscle potentiation was realised. Implementing pre-conditioning exercises with the goal of hormone elevation could negatively influence subsequent motor performance within athletic groups who demonstrate a range of maximum strength capacities. Testosterone appears more responsive to higher volume oriented exercise due in part to the biological effects of the fatigue by-product lactate (e.g. repeated and one off cycle sprint exercise (Crewther et al., 2011b)], moderate intensity high volume resistance training (McCaulley et al., 2009a, Smilios et al., 2003)]). Consequently, it seems logical to suggest that these training modes be performed in isolation and other strategies which avoid any potential for peripheral muscle fatigue (i.e. non-physical stimulation) are employed to initiate the performance enhancing effects hormones might have on physical performance.

The leg press protocol in Chapter 5 was designed to facilitate muscle potentiation through traditional mechanisms (i.e. phosphorylation of myosin regulatory light chain and elevation in spinal reflex processing), whilst also stimulating a hormonal response. No meaningful improvements or decrements in physical performance were identified. The positive performance effects identified in Chapter 4 as a consequence of complex training were not therefore mirrored in the acute response design of Chapter 5. These findings reflect the importance of programme design specificity for PAP strategies. Whilst previous research into PAP development would indicate the leg press contraction mode (isotonic), intensity (86%

1RM), and recovery prescription (8 minutes), was sufficient for the development of PAP, the performance exercise (counter movement jump) was biomechanically different. Consequently, slight mechanical differences in complex set exercise selection could have reduced the potential for muscle potentiation to be translated during the counter movement jump. This was not the case in Chapter 4, as the back squat to jump squat, and bench press to bench throw complexes proved effective at improving power. Equally, muscle potentiation may not have been realised after performing the single complex set training session which characterised Chapter 5. The improvements in power observed in response to chronic complex training in Chapter 4 may suggest the efficacy of pre-conditioning exercise requires multiple training sessions before any beneficial effects are realised in an applied environment.

The final experimental study investigated methods for manipulating the programme design factors of HIT to support repeated power production under conditions of fatigue. The protocols which used shorter work intervals increased the recruitment of selected fast motor units potentially due to the greater contractile demands (i.e. force and velocity requirements) of the exercise design. If a HIT session requires an athlete to generate greater mean and peak power more frequently, then it is assumed that an elevated adaptation potential (i.e. improved repeated power capacity) could occur in Type II muscle fibres. This theory needs to be tested over chronic training periods, but Chapter 6 demonstrated how small changes in the duration of work performed during each interval can initiate meaningful differences in the total amount of high power work performed over an acute training session. This study also demonstrated, for the first time in an applied elite rugby environment, a non-invasive, effective and practical methodological approach using SEMG for investigating the recruitment potential of specific muscle fibres in response to a high intensity exercise stimulus.

The information from this study will be useful to strength and conditioning coaches looking to manipulate the intensity of HIT over repeated sessions to ensure progressive overload is achieved. It also supports the use of cycle HIT as an effective alternative to traditional running with changes of direction. Cycle ergometer training is employed in elite team sport environments for two primary reasons; i) as a mean of providing a high-intensity training stimulus without the musculoskeletal loading associated with high speed running with changes of direction, and ii) during rehabilitation from injury, when a conditioning stimulus is required but the athlete is unable to run at sufficient velocity. Consequently, manipulating the programme design of cycle HIT through reduced work duration intervals could provide a useful method of increasing training intensity for elite team sport athletes during reconditioning phases when off-feet training is preferred.

7.2 Methodological Considerations

The experimental designs of the four studies were implemented and delivered within the complexities of a real elite sport setting and therefore incorporate the normal training constraints inherent to professional rugby players during pre and in-season periods. Factors such as athlete genetics, fatigue, injury status, motivation and training adherence could have influenced the physical performance responses identified throughout the multiple yearly time points assessed in this thesis.

For example, Chapters 4 and 5 demonstrated inter-individual variability in both hormonal, strength and power response patterns to an acute and chronic training stimulus which may reflect the genetic variation which characterise elite rugby groups (Heffernan et al., 2015). The varying levels of neuromuscular capacity in rugby union players reflects the different athletic demands of each playing position but will also influence the strength and power development potential to a given training stimulus.

The data collected during in-season training phases includes the impact that fatigue may have on the athlete's potential for physical development. Over long in-season training periods the potential for strength and power adaptation and expression can be limited by fatigue, fatigue is also associated with increased injury risk (Gabbett, 2005). Fatigue can limit an athlete's ability to adapt maximally to a training stimulus (Gabbett, 2005), whilst injury can limit an athlete's potential to participate in training sessions (Williams et al., 2013). Both these factors would have influenced the current athletic groups potential to respond to the acute and chronic training programmes administered in Chapters 3, 4, 5 and 6.

Athlete motivation to perform the training protocols prescribed in this thesis could also have influenced the training outcomes. Chapter 5 and Chapter 6 did not blind the participants to the nature of the training protocols due to their familiarity with both complex and HIT exercise structures, respectively. As a consequence, any observed performance improvements or decrements witnessed in response to either pre-conditioning stimulus may be associated with the preconceived expectation (positive or negative) an athlete may have regarding PAP strategies (i.e. placebo effect). Equally, each participant had full knowledge and previous experience of short HIT exercise prior to the commencement of each exercise strategy. The greater power in response to the shorter HIT protocols could therefore have been influenced by pre-selected pacing strategies. The acute exercise protocols administered in Chapter 5 were also not subsequently repeated. It cannot therefore be discounted that the individual

performance responses identified were due, in part, to natural variation rather than differences in physical capacities (i.e. strength status).

Whilst differences between the complex and strength training interventions were realised in Chapter 4, the short training periods (4 weeks) may have limited the identification of the effectiveness of one training programme over the other. Complex training studies over longer intervention periods may therefore be required to assess the impact of this training mode not only on power but also its chronic effects on strength development, maintenance and decay. However, the training time frames in Chapter 4 reflect realistic periods over which strength and power cycles are administered within elite team sport athletes competing on a weekly basis.

Any positive and or negative performance responses resulting from the chronic and acute complex (Chapter 4 and Chapter 5) and HIT (Chapter 6) training studies should also be interpreted with respect to the training status (e.g. elite rugby athletes) and athletic environment (e.g. professional rugby team) used for analysis. It is not known whether other athletes or sports teams would exhibit the same training responses and this should be considered when extrapolating the results from this thesis.

7.3 Future research directions

The studies in this thesis have provided a deeper insight into training methods which facilitate strength and power development during pre and in-season training phases. These findings have also highlighted potential avenues for future research that may continue to develop our understanding of how strength and power qualities can be trained in conjunction with the demands of a professional rugby environment.

Future long-term monitoring into the effects of a competitive season on strength and power in rugby players competing in the English premiership should include the impact of the off-season. This period usually lasts for four weeks and athletes are encouraged to rest and regenerate after the rigours of a long competition period. Monitoring the detraining effects of the off-season on strength, power and body composition is of great interest to strength and conditioning coaches. Knowledge of the magnitude in physical decay will help inform the prescription of training loads and goal setting during the initial phases of pre-season. Monitoring off-season detraining may also provide practitioners with information as to the physical capacities most susceptible to regression during periods of inactivity. This knowledge may lead to practical recommendations as to the types of training (e.g. strength, power, speed

or HIT), and the frequency of training required during the off-season to ensure physical qualities are retained for the commencement of a new pre-season training phase.

The scope and magnitude for strength and power development in elite rugby athletes performing in the southern hemisphere has been reported to diminish over multiple seasons (Appleby et al., 2012, Baker, 2013). It would therefore be of interest to extend the current model of longitudinal tracking of English premiership rugby players over multiple seasons to examine how the magnitude of strength and power development evolves in conjunction with increased training exposure and history. The tracking study in this thesis also analysed strength and power adaptation in the lower body; future investigations could include upper body measures as well as specific performance measures such as sprint momentum to further expand our understanding of how rugby athletes develop and translate force over the multiple phases of a full season. Future analysis should also look to monitor academy athletes involved in the English premiership in order to identify a long term athletic development model which benchmarks physical performance requirements during the important training stages of a professional career (e.g. training to compete and training to win stages) (Baker, 2013).

Research from this thesis supports a role for a block period of complex training in support of strength and power. The intervention used reflected the importance of specificity in terms of programming similar movement patterns between complexes and the need to include speed training if improvements in this variable are to be produced. Research needs to be conducted into the design factors which support complex training as a method for potentiating speed. This may be achieved through the assessment of heavy sled acceleration training as a complex with free acceleration training. Acute research could primarily be conducted into the load (i.e. weight on sled) and recovery prescription characteristics of the heavy pre-conditioning sled acceleration. This may be followed by chronic training analysis to assess the impact of sprint performance across multiple complex training exposures.

The unpredictable nature of chronic hormone monitoring observed in this research programme limits any recommendations which can be made as to the value of performing future research focussed on tracking hormones and training adaptation in elite rugby environments. However, this thesis did not assess the potential role of acute testosterone elevation and performance potentiation in response to non-physical pre-conditioning routines. This method has been used successfully to improve the acute expression of maximum strength in an applied training environment (Cook and Crewther, 2012); however, it has yet to be validated in measures of dynamic performance and as a valid strategy for elevating training adaptation over repeat

exposures. This could be a viable area for applied future research as implementing visual strategies (e.g. aggressive or motivational video footage) could easily be delivered to team sport athletes at either a group or individual level immediately prior to the commencement of strength and power training sessions.

The effectiveness of short HIT protocols to initiate fast muscle activation and heightened acute power responses could be analysed over chronic training periods. Whilst intensity appears to be modifiable during HIT through decreasing work interval durations, future research must identify if greater training intensity and fast muscle activation translate to elevated intrinsic adaptation in type II muscle fibre. This may be achieved through pre to post exercise monitoring of intracellular markers within specific muscle fibres that indicate training adaptation (e.g. mitochondrial biogenesis gene expression), in conjunction with SEMG recruitment measurements (e.g. wavelet analysis), and markers of muscle performance (e.g. power). This type of further research may help validate this training tool as an effective means of improving repeated power production through exercise mediated adaptation in fast muscle fibre.

7.4 Practical applications

This research was designed to enhance training prescription for strength and conditioning coaches working within elite rugby union environments. The principal applications of this thesis will be divided into; i) longitudinal applications, ii) acute training applications, and iii) chronic training applications, for the development of strength and power.

7.4.1 Longitudinal applications

- The information from Chapter 3 has provided a unique understanding of the pre and in-season adaptation potential and programme prescription characteristics of rugby players competing in the English premiership. The periodisation structure and adaptive outcomes realised in Chapter 3 may however be highly specific to the environment and athletes used for analysis. Nevertheless, it can be inferred from Chapter 3 that lower body strength and power training can be improved during pre-season and early to mid-season competition cycles. During this period, approximately 1.5 lower body resistance training sessions per week performed with periodised blocks of; i) high load volume hypertrophy training (e.g. 4-6 sets, 4-10 reps at 75-89% 1RM) with the inclusion of accentuated eccentric exercises, and ii) maximal strength training (e.g. 4-6

sets, 2-4 reps at 89-96% 1RM), can be effective at increasing peak force. Approximately 1 lower body speed-power training session per week performed with weighted and un-weighted jumping exercises in conjunction with resisted and un-resisted sprinting may also be effective at increasing maximum power.

- Lower body strength and power can be maintained during end of season cycles. During this period, approximately 1 lower body resistance training session per week performed with reduced load volume hypertrophy and maximum strength training (as utilised in Chapter 3) with speed-power training performed every second week, was sufficient to avoid strength and power regression within the structure of the current environment.

7.4.2 Acute training applications

- Chapter 5 demonstrates that acute muscle potentiation from a pre-conditioning exercise routine may be dependent on athlete specific characteristics such as maximum strength. For a complex set to optimal, it is recommended that programme design factors such as strength, exercise intensity, volume, and PAP recovery interval duration be developed and prescribed on an individual basis.
- Focussing physical exercise strategies on modifying free-testosterone levels could negatively influence an athlete's balance between muscle fatigue and potentiation and as a consequence reduce performance. This method for promoting the non-genomic effects of hormones on muscle performance should be approached with caution.
- For strength and conditioning coaches looking to increase total power and fast muscle activation responses to a cycle HIT session, it is recommended that protocols matched for total exercise volume be constructed with shorter (10 or 15s) work and relief (20 or 30 s) interval durations.
- The use of SEMG derived estimates of muscle activation through wavelet analysis techniques is a novel and practical method for identifying the recruitment capacity of specific muscle fibre types in response to a cycle HIT exercise protocol. This analysis method can be used in the applied setting for discerning the impact of training intensity on muscle fibre specific adaptation potential to cycle sprint training modalities.

7.4.3 Chronic training applications

- For complex training to be effective at maintaining strength and increasing power, programme design factors may incorporate high intensity (83-92% 1RM), low volume (3-4 sets, 3-6 reps), strength exercise prescription alongside moderate intensity power (40-60% 1RM) and plyometric exercise prescription. Strength and power complex sets can be selected based on biomechanical similarities (e.g. back squat to jump squat, bench press to bench throw).
- Based on the recovery intervals utilised in Chapter 4, four minutes rest between strength (pre-conditioning exercise) and power exercises may be sufficient to facilitate the improvements in power in response to a chronic complex training programme.
- Complex training can support power development over short time frames during periods of the year when strength and power training frequency may be limited. It is therefore an effective in-season training tool. Complex training could also provide training variation into an athlete's in-season physical development programme that may reduce training monotony.
- In response to the training structure presented in Chapter 4, it is recommended that complex training be used for short cycles (i.e. 4 weeks), as long-term usage could reduce the adaptation potential for strength development. As a relationship exists between strength and power (as demonstrated in Chapter 3), prolonged complex training cycles could limit power development due to lower magnitudes of strength improvement.
- The lack of reproducibility in acute and chronic hormone response patterns limits the recommendations that can be made as to the efficacy of hormone monitoring for the purpose of identifying markers of training adaptation. As hormone sampling and analysis is time consuming and costly, it is suggested that sports scientists and strength and conditioning coaches in professional rugby environments assess training efficacy primarily through physical performance measures. More research in a professional rugby setting is required before practically meaningful recommendations can be derived as to value of hormones for monitoring training and performance.

7.5 Thesis Conclusion

This thesis has addressed strength and power in an applied context with a focus on three areas: 1) longitudinal tracking, 2) acute training responses, and 3) chronic training responses. The

subsequent research outcomes have provided practical information which expands the knowledge and methods available for monitoring and developing strength and power qualities in professional rugby union players in the English premiership. Initial study demonstrated for the first time that strength and power characteristics can be improved in response to a full English premiership rugby union season and that physical development must be targeted during pre, early and mid-season phases. It was then confirmed that complex training has a positive impact on power development during periods of a competitive rugby season when physical training opportunities are restricted. Studies taking a chronic and acute perspective demonstrated that testosterone and cortisol represent unpredictable markers of training efficacy which makes the practical relevance of this monitoring method difficult to clarify. Lastly, a novel SEMG technique was utilised within an applied setting to demonstrate that manipulating programme design characteristics does influence specific muscle and performance responses to a high intensity training protocol.

The participants in this thesis were elite rugby players, however, coaches, sports scientists, and athletes performing within similar time limited concurrent performance structures should be able to extrapolate relevant information for the applied development of training methods to promote adaptation in strength, power and repeated power production.

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APPENDICES

Appendix 1. Informed consent and participant information forms.

Appendix 1a. Changes in strength and power in professional rugby union players over a playing and training season.

Informed consent

Study Title: Changes in strength and power in professional rugby union players over a playing and training season.

I have been provided with information concerning the above named study and have satisfactorily discussed any queries about participation with Mr. Ed Gannon (University of Bath).

I understand that by consenting to participate in this study, I will be asked to:

- Attend six athletic performance testing sessions over a full professional rugby union season.
- Testing will include;
    - A maximal voluntary isometric contraction where peak force will be measured.
    - A maximal jump squat test for average mechanical power output.
    - A 10 metre acceleration test.

I can confirm that;

- I have been advised of the potential risks and burdens associated with this research.
- I understand that my participation in this research is voluntary, that I am free to refuse to participate or withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my relationship with the Leicester Tigers RFC.
- I understand that this study has been reviewed for ethical consideration and approved by the School for Health Research Ethics Approval Panel. If I have any enquires about

the research, I can contact Mr. Ed Gannon (07920011766, ed.gannon@tigers.co.uk). For any concerns or complaints regarding the way in which the research is or has been conducted, I am aware I can contact the School for Health Research Ethics Approval Panel (Secretary to SREAP, School Administrator, School for Health, University of Bath, Claverton Down, Bath, BA2 7AY) in the first instance.

By signing below I am indicating that I consent to participate in the research project entitled "Changes in athletic performance in professional rugby players over a playing and training season" as it has been described to me in an information sheet and in discussions with Mr. Ed Gannon. I understand that the data collected from my participation will be used for journal publications and conference presentations, and I consent for it to be used in that manner.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Researcher's name ……Ed Gannon……

Researcher's Signature: …  …….. Date: 22/4/2013

Photography and video

I provide my consent for the research team to use any photographs or videos of my person for written or oral presentations such as journal articles, conference presentation and reports, ensuring my anonymity is preserved at all times.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Participant information

Project Title: Changes in strength and power in professional rugby union players over a playing and training season.

Researchers: Mr Ed Gannon, Dr Grant Trewartha and Dr Keith Stokes

Study Purpose: The intention of this study is to identify changes in strength, power and speed in professional rugby players across a full training and playing season. Identifying the nature, scope and magnitude of change in athletic performance across season long training and competition cycles may develop a greater understanding into long term training practices for elite rugby players.

Background: Maximising long term development of strength, power and speed is a primary outcome for elite rugby players. However, much of what we know about muscular adaptation to resistance training comes from short term investigations involving athletes with limited training experience. Subsequently, adaptations stemming from short term studies may not reflect adaptations which occur in professional environments where concurrent training structures and high pre-existing strength capabilities limit physical development. Currently, very little longitudinal data exists concerning the extent, nature and magnitude of muscular adaptation during a season long period which incorporates different training phases (pre-season, in-season, off-season) in professional rugby union. Developing a greater understanding into the effectiveness of training protocols during pre-season, in-season and off-season periods will help further our understanding into the scope for adaptation and methodology of training practice within a professional team sport environment.

Methods: To examine the extent, nature and magnitude of change in strength, power and speed in professional rugby players, three testing blocks will be conducted over the course of a professional season. These testing blocks will provide information pertinent to changes in athletic performance as a result of resistance training across pre-season, in-season and off-season training phases. The training programme will be periodised throughout the year. To assess how variation in training load impacts on athletic performance the average weekly frequency of strength, power, speed, conditioning and skills sessions and the number of matches played will be recorded over each training phase. During the yearly cycle, training block 1 will represent the off-season, pre-season and pre-competition phase of the 2013-2014 season (14 weeks). Training block 2 will represent the in-season phase of the 2013-2014 season (43 weeks). Training block three will represent the off-season, pre-season and pre-

competition phase of the 2014-2015 season (14 weeks). The athletic test battery will be performed six times over the course of the three training blocks. Testing will consist of strength, power and speed. Testing for strength will be performed via an isometric squat test at a knee angle of approximately 140˚. Force produced during the squat will be recorded by a portable force platform with measures of peak force used for further analysis. Testing for power will be performed via a jump squat performed in a customised smith machine with a load relative to 180% of the group's average body mass. Average mechanical power will be recorded via an optical encoder analysis system. Speed will be tested via a10 metre acceleration, with times recorded through a timing gate measurement system.

What is required of you? You will be asked to attend six testing sessions over the course of two pre-season and one in-season training blocks. Each testing session will be performed at the same time of day (10 am ± 1 hour).

You will be asked to execute an athletic test battery to assess alterations in performance over the six testing sessions. Tests will include;

- A maximal voluntary isometric contraction where peak force will be measured.
- A maximal jump squat test for average mechanical power output.
- A 10 metre acceleration test.

What risks are involved? None of the measurement techniques involved are invasive and should not present any risk to you.

Freedom of consent: Participation in this study is entirely voluntary. You are free to deny consent before, during or after data collection. Your participation and/or withdrawal of consent will not influence your present and/or future involvement with Leicester Tigers Rugby Club and will have no bearing on your professional future. Your rights to withdraw shall be preserved over and above the goals of the study.

Confidentiality: All questions, answers and results of this study will be treated with absolute confidentiality. In any manuscripts, reports or publications resulting from this study, subject codes rather than names will be used. Therefore, you will not be able to be identified in these reports and publications.

Data and results: Upon completion, all data collected during this study will be retained in a secure place for at least five years so as to comply with the
University's Code of Practice – Research. Your results will be made available to you at the completion of the study.

Enquiries: Should you have any further queries about this study or wish to register an interest, please contact Ed Gannon (07920011766; ed.gannon@tigers.co.uk).

Appendix 1b. Effects of a combined strength and power (complex) training intervention on athletic performance and hormone concentrations in elite rugby union players during an in-season period

Informed consent

Study Title: Effects of a combined strength and power (complex) training intervention on athletic performance and hormone concentrations in elite rugby union players during an in-season period.

I have been provided with information concerning the above named study and have satisfactorily discussed any queries about participation with Mr. Ed Gannon (University of Bath).

I understand that by consenting to participate in this study, I will be asked to:

- Attend two resistance sessions a week for eight weeks
- Perform strength and power based exercises of intensities varying from 79% to 89%
- Have salivary samples taken before and after each training session
- Attend four separate testing blocks
- Perform anthropometric, strength and power based measurements

I have been advised of the potential risks and burdens associated with this research. I understand that my participation in this research is voluntary, that I am free to refuse to

participate or withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my relationship with the Leicester Tigers RFC. I understand that this study has been reviewed for ethical consideration and approved by the School for Health Research Ethics Approval Panel. If I have any enquires about the research, I can contact Mr. Ed Gannon (07920011766, ed.gannon@tigers.co.uk). For any concerns or complaints regarding the way in which the research is or has been conducted, I am aware I can contact the School for Health Research Ethics Approval Panel (Secretary to SREAP, School Administrator, School for Health, University of Bath, Claverton Down, Bath, BA2 7AY) in the first instance.

By signing below I am indicating that I consent to participate in the research project entitled "Effect of a Specific Combined Strength and Power Intervention on Markers of Performance" as it has been described to me in an information sheet and in discussions with Mr. Ed Gannon. I understand that the data collected from my participation will be used for journal publications and conference presentations, and I consent for it to be used in that manner.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Researcher's name: Edward Gannon……

Researcher's Signature: …  …….. Date: 26/2/2010

Photography and video

I provide my consent for the research team to use any photographs or videos of my person for written or oral presentations such as journal articles, conference presentation and reports, ensuring my anonymity is preserved at all times.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Participant information

Project Title: Effects of a combined strength and power (complex) training intervention on athletic performance and hormone concentrations in elite rugby union players during an in-season period

Researchers: Mr Ed Gannon, Dr Grant Trewartha and Dr Keith Stokes

Study Purpose: The intention of this study is to identify whether a combined strength and power training intervention provides an appropriate stimulus to initiate specific strength and power adaptations in the muscular system. This study will also look to ascertain whether enhancements in strength and power measures are mediated via a hormonal response.

Background: The endocrine (hormone) system plays an important role in initiating muscle remodelling. Developing an appropriate stimulus which acutely elevates the hormonal environment is vital for the development of muscular hypertrophy, strength and power. Factors such as sex, age and nutrition have all been reported to impact upon the hormonal environment. However, a factor which may influence the endocrine response (and therefore the muscular adaptations observed) may be the type of resistance training undertaken. Identifying acute hormone responses to weight training protocols has therefore become a popular area of research.

To date, research has primarily focused on hormonal responses to high volume training schemes which utilise lower training intensities. Whilst these protocols have proved effective at initiating muscle growth through hormonal pathways, many athletes require more selective muscular adaptations. These may primarily be achieved through higher intensity resistance protocols, which require large force development combined with dynamic muscular contractions. By identifying the role the endocrine system plays in response to strength and power activities, a better understanding may be gained as to the mediating role the hormonal system plays at developing markers of performance across the force-velocity continuum.

Methods: Participants will perform two resistance training interventions. Each protocol will be performed over a four week block to ensure all participants execute both interventions. A two week washout period, where no training will occur, will separate the two training blocks. A battery of anthropometric and physical tests will be performed prior to and following each intervention block. Acute hormonal responses to the different protocols will also analysed. These will then be correlated to the various anthropometric and performance based adaptations which have occurred as a result of each training scheme.

What is required of you? You are required to attend two gym-based training sessions per week on non-consecutive days for the eight week intervention period. You will be required to provide a salivary sample before and after each training session. Each weekly session will occur at the same time of the day and will involve a general warm up followed by whole body strength and power based exercises.

You will also be required to attend four testing sessions. Each testing session will involve anthropometric measures for body mass and percentage body fat. You will also be asked to perform a test battery which will include;

1. Three maximum sprints over 20m.
2. Five jump squats and five bench throws with a load corresponding to 55% of your maximum.
3. A maximum strength test, where you will be required to perform a squat and bench press exercise at 86% of your maximum.

Sufficient rest will be given between each performance test and between each contraction to allow recovery and prevent fatigue.

What risks are involved? None of the measurement techniques involved are invasive and should not present any risk to you above your normal weight training. The maximal and near maximal exertion required by some contractions during testing and training may result in mild discomfort.

Freedom of consent: Participation in this study is entirely voluntary. You are free to deny consent before, during or after data collection. Your participation and/or withdrawal of consent will not influence your present and/or future involvement with Leicester Tigers Rugby Club

and will have no bearing on your professional future. Your rights to withdraw shall be preserved over and above the goals of the study.

Confidentiality: All questions, answers and results of this study will be treated with absolute confidentiality. In any manuscripts, reports or publications resulting from this study, subject codes rather than names will be used. Therefore, you will not be able to be identified in these reports and publications.

Data and results: Upon completion, all data collected during this study will be retained in a secure place for at least five years so as to comply with the
University's Code of Practice – Research. Your results will be made available to you at the completion of the study.

Enquiries: Should you have any further queries about this study or wish to register an interest, please contact Ed Gannon (07920011766; ed.gannon@tigers.co.uk).

Appendix 1c. Short-term effects of a weightlifting and cycle sprint warm-up protocol on hormonal responses and power development.

Informed Consent

Study Title: Short-term effects of a weightlifting and cycle sprint warm-up protocol on hormonal responses and power development.

I have been provided with information concerning the above named study and have satisfactorily discussed any queries about participation with Mr. Ed Gannon (University of Bath).

I understand that by consenting to participate in this study, I will be asked to:

- Attend one familiarisation and three testing sessions over a four week period.
- Perform a leg strength training exercise with intensities around 86% 1 repetition maximum
- Perform a maximum cycle sprint protocol

- Perform a battery of dynamic neuromuscular assessments
- Have blood samples taken via fingertip before the leg strength and cycle sprint protocol and before the neuromuscular test battery.

I have been advised of the potential risks and burdens associated with this research. I understand that my participation in this research is voluntary, that I am free to refuse to participate or withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my relationship with the Leicester Tigers RFC. I understand that this study has been reviewed for ethical consideration and approved by the School for Health Research Ethics Approval Panel. If I have any enquires about the research, I can contact Mr. Ed Gannon (07920011766, ed.gannon@tigers.co.uk). For any concerns or complaints regarding the way in which the research is or has been conducted, I am aware I can contact the School for Health Research Ethics Approval Panel (Secretary to SREAP, School Administrator, School for Health, University of Bath, Claverton Down, Bath, BA2 7AY) in the first instance.

By signing below I am indicating that I consent to participate in the research project entitled "The effects of a weightlifting and cycle sprint protocol on hormones and power" as it has been described to me in an information sheet and in discussions with Mr. Ed Gannon. I understand that the data collected from my participation will be used for journal publications and conference presentations, and I consent for it to be used in that manner.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Researcher's name ……Ed Gannon……

Researcher's Signature: …  …….. Date: 24/10/2011

Photography and video

I provide my consent for the research team to use any photographs or videos of my person for written or oral presentations such as journal articles, conference presentation and reports, ensuring my anonymity is preserved at all times.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Participant information

Project Title: Short-term effects of a weightlifting and cycle sprint warm-up protocol on hormonal responses and power development.

Researchers: Mr Ed Gannon, Dr Grant Trewartha and Dr Keith Stokes

Study Purpose: The intention of this study is to identify whether two separate lower body pre-conditioning exercises can lead to enhancements in dynamic lower and upper body neuromuscular performance. This study will also look to analyse the acute hormonal responses to the two pre-conditioning exercises and identify if associations exist between hormonal response and alterations in neuromuscular performance.

Background: Current evidence suggests the initiation of post activation potential (PAP) provides a potent stimulus for enhanced dynamic neuromuscular performance in trained athletes. Pre-conditioning maximal isometric and weightlifting activities have been shown to initiate the heightened neural and muscular response associated with PAP. Subsequently it has been suggested that training sessions focused on power development may benefit from the initiation of a PAP stimulus.

Changes in testosterone and cortisol have also been reported to moderate short term enhancements in neuromuscular function. Resistance training and cycle sprint protocols have both reported acute elevations in endogenous hormone concentrations. These elevations may have implications for neuromuscular performance via an enhanced PAP response. Current evidence examining the role of exercise induced increases in hormonal concentrations on neuromuscular performance remains limited. By analysing hormonal and dynamic

neuromuscular responses to a weight training and cycle sprint protocol, a greater understanding into the associations between hormonal response and PAP development may be gained.

Methods: Participants will perform three testing and one familiarisation session. The familiarisation session will be performed first and will involve leg press and bench press 1 repetition maximum testing followed by practice of the neuromuscular test battery. Each testing session will be performed over a three week period. Testing session one will involve the execution of a leg press pre-conditioning protocol. After eight minutes recovery participants will perform the neuromuscular test battery. Testing session two will involve the execution of a cycle sprint pre-conditioning protocol. After eight minutes recovery participants will perform the neuromuscular test battery. Testing session three will serve as the control and participants will only be required to perform the neuromuscular test battery. Blood samples for acute hormonal analysis will be collected before each pre-conditioning activity in testing sessions one and two, followed by a second sample immediately prior to the execution of the neuromuscular test battery. In testing session three blood sampling will take place immediately prior to the neuromuscular test battery. Neuromuscular responses to both the leg press and cycle sprint protocols will be analysed alongside potential acute alterations in hormonal response.

What is required of you? You will be asked to attend an initial familiarisation session, followed one week later by three testing sessions. Each testing session will be performed at the same time of day separated by one week. You will be required to provide blood samples via fingertip before each pre-conditioning exercise and prior to each neuromuscular test battery.

You will be asked to execute a test battery to assess alterations in neuromuscular performance, tests will include;

1. A smith machine jump squat on a portable force platform with an absolute load of 18 kg. Measures of peak ground reaction force, peak rate of force development, peak power output and jump height will be recorded.
2. A smith machine bench throw with an absolute load equivalent to 40% of the group mean 1 repetition maximum bench press. Measures of peak power output will be recorded.

What risks are involved? None of the measurement techniques involved are invasive and should not present any risk to you. Slight discomfort may be experienced when blood sample is obtained via fingertip.

Freedom of consent: Participation in this study is entirely voluntary. You are free to deny consent before, during or after data collection. Your participation and/or withdrawal of consent will not influence your present and/or future involvement with Leicester Tigers Rugby Club and will have no bearing on your professional future. Your rights to withdraw shall be preserved over and above the goals of the study.

Confidentiality: All questions, answers and results of this study will be treated with absolute confidentiality. In any manuscripts, reports or publications resulting from this study, subject codes rather than names will be used. Therefore, you will not be able to be identified in these reports and publications.

Data and results: Upon completion, all data collected during this study will be retained in a secure place for at least five years so as to comply with the
University's Code of Practice – Research. Your results will be made available to you at the completion of the study.

Enquiries: Should you have any further queries about this study or wish to register an interest, please contact Ed Gannon (07920011766; ed.gannon@tigers.co.uk).

Appendix 1d. The effect of work interval duration on power and muscle fibre activation during high-intensity interval training (HIT).

Informed Consent

Study Title: The effect of work interval duration on power and muscle fibre activation during high-intensity interval training (HIT).

I have been provided with information concerning the above named study and have satisfactorily discussed any queries about participation with Mr. Ed Gannon (University of Bath).

I understand that by consenting to participate in this study, I will be asked to:

- Attend three HIT testing sessions over a three week testing period
- Testing will include;
    - A maximal voluntary cycle sprint lasting 5 seconds followed (after four minutes of recovery) by a HIT protocol incorporating 24 reps of 10 second cycle sprints interspersed with 20 second rest intervals
    - A maximal voluntary cycle sprint lasting 5 seconds followed (after four minutes of recovery) by a HIT protocol incorporating 16 reps of 15 second cycle sprints interspersed with 30 second rest intervals
    - A maximal voluntary cycle sprint lasting 5 seconds followed (after four minutes of recovery) by a HIT protocol incorporating 12 reps of 20 second cycle sprints interspersed with 40 second rest intervals

I can confirm that;

- I have been advised of the potential risks and burdens associated with this research.
- I understand that my participation in this research is voluntary, that I am free to refuse to participate or withdraw from the research at any time. My refusal to participate or withdrawal of consent will not affect my relationship with the Leicester Tigers RFC.
- I understand that this study has been reviewed for ethical consideration and approved by the Research Ethics Approval Committee for Health (REACH). If I have any enquires about the research, I can contact Mr. Ed Gannon (07920011766, ed.gannon@tigers.co.uk). For any concerns or complaints regarding the way in which the research is or has been conducted, I am aware I can contact the Research Ethics Approval Committee for Health (Secretary to SREAP, School Administrator, Department for Health, University of Bath, Claverton Down, Bath, BA2 7AY) in the first instance.

By signing below I am indicating that I consent to participate in the research project entitled**:** **“**The effect of work interval duration on power and motor unit recruitment during high

intensity interval training (HIT)." as it has been described to me in an information sheet and in discussions with Mr. Ed Gannon. I understand that the data collected from my participation will be used for journal publications and conference presentations, and I consent for it to be used in that manner.

Participant's name ……

Participant's Signature: ..... Date: ...../……../..…..

Researcher's name ……Ed Gannon……

Researcher's Signature:  …….. Date: .....28/3/2014.....

Photography and video

I provide my consent for the research team to use any photographs or videos of my person for written or oral presentations such as journal articles, conference presentation and reports, ensuring my anonymity is preserved at all times.

Participant Information

Project Title: The effect of work interval duration on power and muscle fibre activation during high-intensity interval training (HIT).

Researchers:

Mr Ed Gannon; ed.gannon@tigers.co.uk 07920011766
Dr Grant Trewartha; g.trewartha@bath.ac.uk 01225383055
Dr Keith Stokes; k.stokes@bath.ac.uk 01225384190

Study Purpose: You are invited to take part in a study that will assess the effect of manipulating the work interval duration on muscle fibre type activation and power output during high intensity interval training (HIT) exercise strategies. The information gained will

shape guidelines regarding the work interval duration that provides the greatest engagement of type II muscle fibres.

Before deciding whether to take part, it is important that you understand why the study is being done and what you are being asked to do. Take time to read the following information carefully and if there are any aspects of the study that you do not understand, please discuss them with one of the investigators. When you have read and fully understood the information, if you decide that you would like to take part you will be asked to sign a consent form.

Background to the study: Rugby players are required to repeatedly produce high force efforts interspersed with brief recovery intervals over an extended period of time. Subsequently, performance relies not only on an individual's absolute force production capacity, but also on their ability to reproduce high power outputs under conditions of fatigue. High intensity interval training (HIT) uses repeated short bouts of high intensity exercise interspersed with short recovery intervals. HIT exercise protocols allow for large cardiac outputs and motor unit recruitment (i.e. type II muscle fibre) to amplify adaptation. The potential for long-term physiological development and the time efficient nature of HIT make this mode of exercise popular within elite team sport environments. However, several exercise programming factors must be considered to ensure a given HIT protocol is delivered optimally, specifically in relation to the time durations assigned to work and rest periods, but this information is currently lacking.

What will I have to do? You will be asked to attend three testing sessions over a three-week period during the in-season phase of professional rugby season. Each testing session will be performed at the same time of day (10 am ± 1 hour).

At each testing session you will initially be asked to perform a maximum six-second sprint on a watt bike. After a four-minute recovery period you will be required to perform a HIT cycle protocol. This will require you to cycle at high intensity muscle contractions for a fixed period of work separated by a fixed period of rest. Each HIT protocol you will be asked to perform over the three-week testing period.

What risks are involved? The trials will require you to perform physically intense activities identical to your fitness training and thus the risks of the experiment will be the same as

normal fitness training. None of the measurement techniques involved are invasive and should not present any risk to you.

Freedom of consent: Participation in this study is entirely voluntary. You are free to deny consent before, during or after data collection. Your participation and/or withdrawal of consent will not influence your present and/or future involvement with Leicester Tigers Rugby Club and will have no bearing on your professional future. Your rights to withdraw shall be preserved over and above the goals of the study.

Confidentiality: All questions, answers and results of this study will be treated with absolute confidentiality. In any manuscripts, reports or publications resulting from this study, subject codes rather than names will be used. Therefore, you will not be able to be identified in these reports and publications.

Data and results: Upon completion, all data collected during this study will be retained in a secure place for at least five years so as to comply with the
University's Code of Practice – Research. Your results will be made available to you at the completion of the study.

What are the benefits of involvement?: You will receive a record of your current aerobic and anaerobic capacity. It is envisaged that the study will inform future training practices to enhance development of strength and power in rugby players.

Ethics Approval

This project has been reviewed by and received ethics clearance through the Research Ethics Approval Committee for Health (REACH).

Enquiries: Should you have any further queries about this study or wish to register an interest, please contact Ed Gannon (07920011766; ed.gannon@tigers.co.uk).

Participant's name ……

Participant's Signature: ..... Date: ...../……../……..